



Merry, Karen Jane (2010) *Murder by poison in Scotland during the nineteenth and early twentieth centuries*. PhD thesis.

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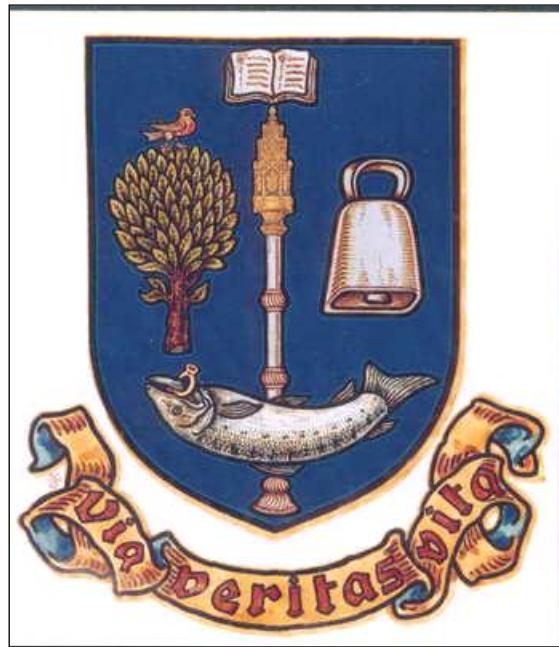
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Murder by Poison in Scotland During the Nineteenth and Early Twentieth Centuries

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ABSTRACT

This thesis examines the history of murder by poison in Scotland during the nineteenth and early twentieth centuries, in the context of the development of the law in relation to the sale and regulation of poisons, and the growth of medical jurisprudence and chemical testing for poisons.

The enquiry focuses on six commonly used poisons. Each chapter is followed by a table of cases and appendices on the relative scientific tests and post-mortem appearances. The various difficulties in testing for these poisons in murder and attempted murder cases during the period are discussed and the verdicts reached by juries in poisoning trials considered.

It is argued that murder by poison during the nineteenth and early twentieth centuries raised particular legal and medical problems, as not only were symptoms often not recognised by doctors, but chemical testing was inadequate, and juries as arbiters of fact often did not understand the evidence that was presented to them in court during trials for poisoning. Further, the ease with which these poisons could be purchased for very small sums of money, the rise of the insurance industry, and the prominence of burial clubs all contributed to providing opportunity and motive for murder.

Since poisons were easy to obtain and difficult to detect, it seems probable that poisoning was much more common than is usually accepted.

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ABBREVIATIONS

AD – Records of The Lord Advocate

BMJ – British Medical Journal

Edinburgh New Phil Journal – Edinburgh New Philosophical Journal

EMJ – Edinburgh Medical Journal

GMJ - Glasgow Medical Journal

HMSO – His (Her) Majesty's Stationary Office

JC – Justiciary Court Records

Med.Leg.Rev – Medical Legal Review

Pharm. J. - Pharmaceutical Journal

PP – British Parliamentary Papers

S.R. & O – Statutory Rules and Orders

Social Stud Science – Social Studies in Science.

Surgo Glasgow University Med J – Surgical Glasgow University Medical Journal

Dedication

If I have written this for anyone in the world then it would be for my dad, Douglas Smith, who all my life has been my silent inspiration. I miss him every day and I dedicate my thesis to his memory.

Additionally, I dedicate my thesis to the memory of my two grandfathers, Harry Gordon Mathieson Laing and Francis Richard Smith who both would have been so very, very proud and thought this a remarkable accomplishment.

“Death leaves a heartache no one can heal,
Love leaves a memory no one can steal.”

(Neil Young)

Acknowledgements

My foremost thanks go to my esteemed PhD supervisor, Professor Olivia Robinson. Her unstinting encouragement, enthusiasm, support and guidance over the years have given me the confidence, determination and strength to complete this thesis and I am deeply indebted to her.

I am also very grateful to the University of Glasgow for granting me extensions for this thesis due to periods of ill health.

Sincere appreciation and gratitude is also extended to the staff at the National Archives of Scotland in Charlotte Square, Edinburgh for all the help I was given with obtaining the records for my research.

Many thanks also go to the Wellcome Trust who provided me with a grant to help fund my numerous trips to Edinburgh.

With warm appreciation and gratitude I wish to acknowledge Dr Paul J McKevinney for his never ending patience, help and advice. Also many thanks also to Dr Graham McMillan whose help has been invaluable to me.

Great many thanks I would also like to give to my friends Sam and Harry for their continued friendship, good company, and fantastic food. Thank you Ben as well.

Thanks also to my aunty Clem and uncle Rob for all their kindness over the years.

Finally, my deepest thanks and love go to my mum, Dorothy Smith, for checking and correcting all the grammar in my thesis and for her continued patience, words of encouragement, support and love. Without her backing I would never have reached this point and in the words of Neil Young, “Your love is like a light showing me the way.....”

DECLARATION

I declare that, this dissertation is the result of my own original research. It has been composed by me and has not been submitted for any other degree at the University of Glasgow or any other institution.

The copyright of this thesis belongs to the author under the terms of the United Kingdom Copyright Acts and due acknowledgement must always be made of the use of any material contained in, or derived from, this thesis.

Signature _____

Printed Name _____

Chapter 1

1.1Introduction

*Sleeping within my orchard,
My custom always of the afternoon,
Upon my secure hour thy uncle stole,
With juice of cursed hebona in a vial,
And in the porches of my ears did pour
The leperous distilment; whose effect
Holds such an enmity with blood of man
That swift as quicksilver it courses through
The natural gates and alleys of the body,
And with a sudden vigour doth posset
And curd, like eager droppings into milk,
The thin and wholesome blood. So it did mine,
And a most instant tetter bark'd about,
Most lazarus-like, with vile and loathsome crust,
All my smooth body.¹*

The mass of literary and cultural usages of the morbid mystique of the poisoner's art as a symbol of the darker sides of human nature², attests to the enduring historical fascination with this most insidious of methods of murder. The intrigue associated with the esoterism of the processes of chemical preparation and the stealth and secrecy of the processes of administration prompted the author's own decisions to

¹ Hamlet Act 1 Scene V.

² Of particular interest are the fairy tales of childhood, which always seemed to use the perfect poison such as in Snow White where a poisoned comb and apple were used. See also Grimm's Fairy Tales – *The Poor Boy in the Grave* in which a pot of poison was kept under the bed and Anderson, H.C., *The Evil King* where the king was poisoned by a mosquito. Not to be forgotten also is Lewis Carroll's, *Alice's Adventures in Wonderland*, where food and drink labelled "poison" acquires powers over the human body far more drastic and more benign than those in Alice's real world. Even in modern times poison is still being used in children's books to create an image of magic and mystery. In particular see Rowling, J.K., *Harry Potter and the Philosopher's Stone*; *Harry Potter and the Chamber of Secrets*; *Harry Potter and the Prisoner of Azkaban*; *Harry Potter and the Goblet of Fire*; *Harry Potter and the Order of the Phoenix*. In addition, is the use of poisons in many of Shakespeare's works such as henbane as the poison for Hamlet's father (Act 1, Scene V), or the witches and Lady Macbeth in *Macbeth*.

design the present enquiry investigating poisoning cases between 1800 and 1913 in her native Scotland whilst also reviewing developments in forensic medicine and the legal regulation of poisons. This long century encompasses a period when cheaply available poisons with common innocuous uses could be easily obtained. Further, the inability of medicine and chemistry to identify the use of poison, allied to new potential for insurance claims made by the family of victims, made poisoning much less rare than has often been claimed. Limiting the case study to this century and to the cases brought before Scottish courts enables a broader investigation of the most common types of poisons used. Chapter by chapter, cases involving six of the poisons most regularly found in criminal cases in Scotland in the nineteenth century are unveiled. These toxicants, in turn, are arsenic, corrosive acids, phosphorous, opium, strychnine and prussic acid. In the course of the following chapters, sixty-three cases of poisoning, of which most were found to be murder, are considered. These cases are set in their socio-economic contexts and the propensities for poisoning cases in the nineteenth century are analysed. The effects and development of legislation, the development of medical knowledge and the scientific evolution of tests for poisons are crucial and primary factors in contextualising the observable patterns of poisoning cases.

Such patterns must be set within a contextual background of the increasing interest (public and legal) in criminal trials for poisoning from the mid nineteenth century onwards. The most prevalent poison in the British Isles in the early nineteenth century was arsenic and following an outbreak of Asiatic cholera in England in 1831³, the symptoms of which resembled those associated with arsenic poisoning, it was realised by the scientific community that new and more reliable tests were required to isolate poisoning cases and bring poisoners to justice.

The Marsh test, which is described in more detail in later sections, was the result of this new desire to isolate a way of proving the presence of arsenic. As Watson notes this new scientific impulse transformed not only scientific and juridicial considerations of arsenic poisoning, but invigorated general public and legal interest

³ Watson, K., *Poisoned Lives: English Poisoners and Their Victims*, Hambledon and London, London, 2004, p2.

in poisonings of all kinds.⁴ In Great Britain as a whole in the mid-nineteenth century, a spate of poisoning trials accompanied new methods of detection. Two-hundred and thirty-nine trials in the UK for murder and attempted murder with poison are recorded in the years 1839 to 1849.⁵ Such a mid-century surge was also visible in cases in Scotland taken by itself.

It is precisely the ease of acquisition and use of poisons which motivated the undertaking of this research and which underpins the central contentions of the thesis. Other forms of murder did not engage my curiosity and murder by poison is a topic which is fairly easily isolated. Poisons were readily available in nineteenth century Scotland since many substances, such as arsenic and oxalic acid, had common household uses⁶. This thesis will highlight how very easy it was to obtain poison and will attempt to prove that the use of poison for murder was common. The measures described in this thesis to control poisonous substances explain why there is no useful comparison with modern times which is possible.

By considering the totality of the records of available Scottish poisoning cases in the long nineteenth century in question, the thesis further illustrates the limits of medic-legal practice in Scotland in the Victorian period. The cases under review highlight several ways in which acts of murder by poisoning would never have reached trial and moreover ways in which many poisoners are likely to have escaped punishment, or received only lenient sentences. The singularities of this thesis are to be found in the full continuum of trials in Scotland, especially focusing on the lesser known cases involving the rural and urban poor. The development of interest in poisoning trials

⁴ Ibid.

⁵ See Parliamentary Papers 1850, XLV: *Returns of the Number of Persons Tried in the UK for Murder and Attempts to Murder by the Administration of Poison. In Each Year 1839-1849 Inclusive*, pp447-453.

⁶ Arsenic was used in household paint in the form of copper arsenite which gave a beautiful green colour and was known as Scheele's green whilst a combination of copper acetate with copper arsenite known as emerald green was used in the manufacture of wallpaper, soap, lampshades, children's toys, candles, soft furnishings and even cake decorations- See Emsley, J., *Elements of Murder*, Oxford University Press, 2005, pp116-120. Oxalic acid was used for domestic cleaning, to polish brass and in particular to remove stains- See Glaister, J., *Medical Jurisprudence and Toxicology*, 12th edn, E. & S. Livingstone Ltd, 1966, p497.

and scientific methods of detection mirrored developments in England. The failure, though, to convict in many Scottish cases was exacerbated by the existence of the Not Proven verdict in Scottish law. Though juries all across the British Isles might have been expected to be unwilling to immediately trust toxicological analysis, and much doubt was publicly cast upon the role of the medical witness, the failure in many cases investigated herein to convict probable murderers was made more likely in Scotland by the jury having the option to declare the case Not Proven. This, in essence summed up legal and public attitudes towards the efforts of the scientific community, as well as granting freedom to many likely murderers.

1.2 Research and Methodological Issues

The research for this thesis was carried out over the course of three years and was based on the primary sources located at the National Archives of Scotland within West Register House, Edinburgh, at Charlotte Square. It is here that records of Scottish crimes are kept from the early nineteenth century onwards in the format of the Records of the Lord Advocate allied with archives of court minute books, trial transcripts, and press cuttings, records of suspicious deaths and returns of capital convictions.⁷ The main source of information for this thesis has been the Records of the Lord Advocate. Mining the entire collection from 1800 – 1913 was necessary to determine whether there were any recorded poisoning cases in each time period covered. For each case discovered, all the boxes of papers relating to that case were pulled in order to extract the relevant information. The research undertaken was both time consuming and difficult, due both to the age of some of the papers, the care that had to be taken whilst reading them and the thoroughness required to gain a full overview of the case profiles for such an extended time period. Some of the cases discovered could be matched with Justiciary Court Records (court minute books) and trial transcripts thus providing further contextual information. Some of the cases were also mentioned in Lord Cockburn's *Circuit Journeys*, 1888, which confirmed much of the knowledge obtained from the Records of the Lord Advocate. Other than the

⁷ To be noted is that returns of capital convictions exist only from 1833-1860 and thereafter were not kept. Full trial transcripts exist from 1888 onwards, suspicious death inquiries from 1895 onwards and press cuttings only from 1902 onwards.

poisons focused upon in the subsequent chapter, most other poisons mentioned in the Records seem only to occur once or twice and are isolated incidents from which little of overall sociological value can be derived.⁸

The presentation of the results of this research is organised with individual chapters devoted to the different poisons used rather in chronological divisions. Making arbitrary splits in the time period would not facilitate evaluative reasoning upon the causes of the popularity of poisoning. Instead, by seeking commonalities in cases of poisoning using the same substance, I intend to show that consistent patterns of usage exist which contribute to explaining the overall character of murder by poisoning in nineteenth century Scotland. Whilst many academic writers suggest and maintain that homicidal poisoning was rare in the nineteenth and early twentieth centuries this study contests that orthodoxy.⁹ The six poison chapters attempt to prove that during the nineteenth and early twentieth centuries poisoning in Scotland was fairly common. There existed few barriers to the possession of poison during this time period; the means of murder were simply there for the taking for those with a motive for their

⁸ See Littlejohn, Harvey, *EMJ*, vol XVII, 1905, pp53-65, Medical Jurisprudence.

⁹ In particular see Crowther, A & White, B., *On Soul and Conscience; The Medical Expert and Crime; 150 Years of Forensic Medicine in Glasgow*, Aberdeen University Press, 1988, p19; Burney, I., *Wellcome News*, issue 20, 1999, p3, The Poison Hunter. Anne Crowther was a senior lecturer and Brenda White was a research fellow in the department of Economic History at the University of Glasgow. Ian Burney was a Wellcome Research fellow at the University of Warwick. See also Smith, F., *Cause of Death*, Orbis Publishing, 1980.

use.¹⁰ Indeed, citizens of the nineteenth and early twentieth centuries had only to walk into their local chemist, druggist, grocer's shop or chandlers to purchase a "death-bringer" for a very small sum of money and with no checks or restrictions on their sale until the mid-Victorian period. Further, the medical profession itself dispensed many toxic preparations, and the use of poisons in household cleaning fluids, paint, artist's materials, vermin killers etc. was virtually uncontrolled.

While the breakdown of the chapters by toxin is necessary to elicit conclusions which relate to the properties of each substance, an overview of the chronological history of the poisoning cases under review reveals a consistency of the presence of poisoning cases in the legal records throughout the period under investigation. The percentage of poisoning cases within the chosen time period may be deconstructed as follows: 1800-1810 = five percent (5%); 1811-1820 = eight percent (8%); 1821-1830 = fourteen percent (14%); 1831- 1840 = eleven percent (11%); 1841- 1850 = thirteen percent (13%); 1851- 1860 = thirteen percent (13%); 1861- 1870 = eleven percent (11%); 1871- 1880 = five percent (5%); 1881- 1890 = six percent (6%); 1891- 1900 = six percent (6%); 1901- 1913 = eight percent (8%).

¹⁰ Until 1851 there was no effective legislative controls whatsoever over the sale of poisons in Great Britain. During preceding years, however, the Registrar General's annual returns had disclosed an increasing number of deaths from poisoning, more than one third of which were due to arsenic. The Arsenic Act of 1851 was, therefore, passed. However there was no clause in this Act restricting the sale of arsenic to chemists and druggists since there was at that time no legal definition of a chemist and druggist – Linstead, H., *Poisons Law*, Pharmaceutical Press, 1936, p3. An Act to regulate the Sale of Poisons and alter and amend the Pharmacy Act of 1852 was introduced in 1868. The 1868 Act added sixteen other poisons, sales of which were to be controlled: Prussic Acid; Cyanides of Potassium; All metallic cyanides; Strychnine; All poisonous vegetable alkaloid and their salts; Aconite; Emeric Tartar; Corrosive Sublimate; Cantharides; Savin and its Oil; Ergot of Rye; Oxalic Acid; Chloroform; Belladonna and its Preparations; Essential Oil of Almonds unless deprived of its Prussic Acid; Opium and all Preparations of Opium or of Poppies. These poisons could not be sold unless the buyer was known to the seller or introduced by some person known to the seller. Further, an entry of sale had to be made in a journal stating date of sale, name and quantity of article sold, and the signature of its purchaser plus a signature of any person who had introduced him/her.

1.3 A Crime of the Poor: Trends and Lacunae in Scholarship

Legal and sociological studies, from the past and present, generally have focused on those cases which became historically notorious because the perpetrators were of relatively high social status. Prominent examples included such as the cases of Madeleine Smith and Dr Pritchard.¹¹ This perception has been further fuelled by the fact, as Spierenburg notes, that poisoning was often a crime associated with the upper echelons of society in the more distant past. Intrigue and rivalry in the courts of the classical world often resulted in poisoning, such as that of the Emperor Claudius. Further, “(T)hree-quarters of all poison victims in the Middle Ages were from the upper classes¹²”. While the preparation of poison and the cost of ingredients were prohibitive in earlier periods, the industrial revolution brought poisonous substances within the reach of even the poorest. Murder by poison in the Victorian period often occurred within marriage due to lack of wealth, and the inability of people to remove themselves legally from unhappy unions.¹³ Also the killing of very young children, particularly with the use of opium products, was also once widespread, as they were often considered to be a burden and were a great expense for parents or single mothers. The rise of the insurance industry led to a motive for murder that is today associated with large sums of money, but began with the local burial clubs in Scotland. These schemes entailed weekly or monthly contributions by families with meagre incomes, who, in light of high infant mortality did not wish to be unable to afford a decent funeral for any children who might die in their infancy. By contributing to the club, expenses for a funeral would be paid out by the members

¹¹ Madeleine Smith stood trial for the murder of Pierre Emile L'Angelier in Glasgow in 1857 and Dr Pritchard murdered both his wife and mother in law with opium and antimony in Glasgow in 1865.

¹² Spierenburg, P., *A History of Murder: Personal Violence in Europe from the Middle Ages to the Present*, Cambridge: Polity Press, 2008, p128.

¹³ Women rarely chose to separate from men in the past, particularly if they had children, because so few either rich or poor could survive without the financial support of their husbands or fathers due to limits on women's ownership of property. Also before the religious Reformation in 1560 there was no full divorce (*a vinculo*) allowing re-marriage in Scotland. From that date the courts began to grant divorces for adultery at common law, basing themselves on Biblical texts. In 1573, desertion became a ground of divorce by statute [James V1, 1st Parl. Ch. 55, 1573] and in 1938 the Divorce (Scotland) Act added cruelty, incurable insanity, sodomy and bestiality- See Leneman, Leah, *Alienated Affections: The Scottish Experience of Divorce and Separation 1684 – 1830*, Edinburgh University Press, 1998, p6.

collectively, avoiding the shame of a pauper's funeral. However, suspicions were aroused by the fact that families joined multiple clubs, and could thus collect big payouts if a child of the family died. Thus motive for wilful neglect or murder was established by the lack of regulation of these cooperative schemes.

Further, domestic servants, once the second largest occupational group in the country, had no practical recourse when ill-treated by their employers. Throughout this thesis it will be demonstrated that poisoning was primarily a crime of the poor and underprivileged, a fact that seems to have been under-emphasised in scholarly treatments of poisoning in the Victorian period. This thesis therefore challenges old assumptions and beliefs in trying to prove that poisoning was common in the past albeit that this is secondary to the more substantive claims made about the development of forensic medical and legal history. Further, it gives a voice to those who would otherwise remain unknown in a history of criminal poisoning in Scotland. As Burney notes, the Victorians themselves "thought they were witnessing a 'rebirth' of the crime of poisoning¹⁴" as society became more civilised and turned away from 'direct' forms of murder. However, this fact has been obscured by focus on high-profile cases, and so the bulk of the primary research herein has therefore been focused on forgotten cases - those cases largely unknown to the Scottish public. Reported statistics from the nineteenth century are notoriously unreliable. By examining the entire corpus of the Lord Advocate's Records, however, as solid a factual base as is possible is provided for the conclusions of the enquiry. To make sweeping statements suggesting that homicidal poisoning in the past was rare without undertaking painstaking research and examining the correlated and hugely important development of forensic medicine and law to justify this is unreasonable.¹⁵ Notable in contradiction to this orthodoxy is the eminent Professor of Forensic medicine at Guy's Hospital, London, Keith Simpson, who opined that "(H)omicide by poison is and was not rare in the past."¹⁶ Further, lack of authentic documents containing the medical

¹⁴ Burney, I., *Poison, Detection and the Victorian Imagination*, Manchester University Press 2006, p19.

¹⁵ See for example note 5.

¹⁶ Taylor, A., *Taylors Medical Jurisprudence and Toxicology*, 12th edn, ed: Simpson, K., Churchill, London, 1965, p79.

facts and investigations related to poisoning trials in our own country convey incorrect impressions in relation to exact figures for poisoning cases.¹⁷

During this research, as intimated earlier, details of cases were uncovered where other poisons had been used such as aconite, antimony, mercury (corrosive sublimate), belladonna, cantharides, tartar emetic and chloroform. These were, however, isolated cases in the Records and so they have been eliminated from this thesis.

Early forensic medicine, unlike that of today, lacked co-ordination between pathologists, serologists and chemists. Further, there was no legislation in place during the early nineteenth century to ensure public enquiry into the causes of sudden deaths.¹⁸ It seems hardly unlikely, therefore, that what may have appeared as an accidental poisoning or illness could in fact have been premeditated murder. Indeed, the circumstantial evidence of the character of daily medical practices adds weight to this contention. Many medical men in the nineteenth and early twentieth centuries carried antidote bags with them as a matter of course. Aside from having to deal with accidental poisoning such as opiate overdosing, this practice suggests more common

¹⁷ See for example Glaister, John, "The Power of Poison", Christopher Johnson, London, 1954.

¹⁸ In 1842 a code of instructions were drawn up by the Lord Advocate, and issued from the Crown Office to every Procurator Fiscal. This was in respect to deaths with suspicious circumstances, death from accident, or cases of sudden death. This code of instructions contained *inter alia* an appendix of fourteen folio pages, furnishing detailed directions to medical men for the making of post-mortem examinations - See MacLagan, D., *The Journal of Jurisprudence*, vol XXII, 1879, pp1-22, Forensic Medicine from a Scottish Point of View. Prior to this in all cases of sudden death, the district constable went to the place where it had occurred. He collected information and then sent off a report to his superintendent who, if he felt that the circumstances raised were suspicious, applied to the nearest medical man, without delay, for a post-mortem examination to be carried out and a report prepared. Upon receiving such a report the superintendent would pass it to the Procurator Fiscal of the appropriate area who would decide if further action was to be taken- Craig, J., *EMJ*, vol 14, 1803, pp15-23, On Medical Evidence in the Preliminary Investigations of Criminal Cases in Scotland.

encounters with poisoning than modern scholarship has envisaged.¹⁹

Through discussion of past forensic toxicology and its history it will become clear just how difficult it was to detect that a person had been poisoned in the period in question. Forensic toxicology was perplexing, inaccurate and unsophisticated. In addition, symptoms of poisoning bore resemblance to many other diseases making diagnosis by doctors virtually impossible. This was particularly the case in rural areas where medical practitioners had no experience of forensic medicine, and were not aware of the significant markers which may have led to the diagnosis of possible poisoning.²⁰ Indeed, as symptoms of many diseases resemble each other, doctors had a particularly difficult time in giving a correct diagnosis. It is almost certainly significant that twenty-two of all the poisoning cases investigated in the following chapters (35% of the total) were discovered in either Edinburgh or Glasgow where the two main medical schools giving instruction in forensic medicine were located.²¹ Further, at the beginning of the nineteenth century most doctors were only learning

¹⁹ Such bags usually contained a stomach pump which was six to eight feet of India rubber tubing, a hypodermic syringe, a bleeding lancet, a tracheotomy knife, a small current battery to supply galvanic shocks to walls of the chest, at least four different emetics to induce vomiting and a careful selection of antidotes such as they were- Blyth, A.W., *Poisons: Their Effects and Detection*, Charles Griffin & Co, London 1884. See also Comrie, J.D., *Black's Medical Dictionary*, A & C Black Ltd, London, 1926, p53, where it is advised that an antidote bag should contain atropine for prussic acid poisoning, chloroform for strychnine poisoning, calaber bean for atropine poisoning and atropine for opium poisoning. See also Hill, G.N., *EMJ*, vol 7, 1810, pp22-23 Diagnosing Poisoning with Reference to the Administration of Antidotes.

²⁰ Symptoms of poisoning in the past bore resemblance to diseases such as: alcoholic intoxication, apoplexy, cholera, consumption (phthisis), diphtheria, gastro-enteritis, hysteria, neuralgia, pneumonia, poliomyelitis, tetanus, tuberculosis, typhoid- Comrie, J.D., *Black's Medical Dictionary*, A & C Black Ltd, London, 1926, pp719-722.

²¹ I have nine poisoning cases in Glasgow and thirteen in Edinburgh within the time period 1800 – 1913 – 22/63 x 100 = 35%. From 1807 until the early 1830s, the University in Edinburgh was the only medical school in Britain to give systematic instruction in forensic medicine. Glasgow University, somewhat slow to follow Edinburgh's example, nevertheless in 1839 introduced forensic medicine as a compulsory part of the medical curriculum. The first chair at Glasgow University was created in 1839, whilst the first in Edinburgh was 1807- Crowther, A and White B., *On Soul and Conscience: The Medical Expert and Crime; 150 Years of Forensic Medicine in Glasgow*, Aberdeen University Press, 1988, p7.

their “trade” through apprenticeships, and early doctors in most cases treated symptoms only, because they did not know the cause of a disease or illness.²²

This thesis will also show that the act of poisoning was associated with both sexes in the past and not just women. As Katherine Watson has commented, perhaps due to the non-violent nature of murder by poisoning: “(M)ost people today automatically assume that the typical...poisoner was a woman²³”. The thesis will also highlight the difficulties faced by Scottish juries in poisoning trials due to the fact that in any poisoning trial it was highly unlikely that there would be direct evidence to be presented before the jury.²⁴ Further, the thesis argues that evidence presented to juries in such trials was usually of such a complex medical nature that it was extremely difficult for the lay persons of the jury to understand.²⁵

paraquat seems to be clear cut

The figure of sixty-three poisoning cases, spanning a period of one hundred and thirteen years, may suggest to many that the numbers of murders and attempted murders by poison in Scotland is very small. Comparison with the English figure of three hundred and fifty-seven cases for the time period 1750-1914 would also suggest that poisoning was not a frequent occurrence in the nineteenth century.²⁶ However, there is little doubt that these figures can hardly be representative of a complete history of poisoning in Scotland or indeed England. Many cases would naturally have been missed due to the diagnostic deficiencies of the period. The absolute certainty, facility and simplicity of operation by which most poisons may now be recognised

²² The most common methods of treatment used by nineteenth century doctors were bleeding (phlebotomy), blistering, plastering, amputation, purging, vomiting and sweating. See Comrie, J.D., *Black's Medical Dictionary*, A & C Black Ltd, London, 1926 for full details of all these treatments.

²³ Watson, K. *Poisoned Lives: English Poisoners and Their Victims*, Hambleton and London, London 2004, pp. xiii.

²⁴ Direct evidence may be defined as evidence which leads directly to proof of a fact or facts in issue. Thus for example the evidence of W to the effect that he saw A hit B is, in an assault case, is direct evidence of that assault- See Walker, A.G. and Walker, N.M.L., *The Law of Evidence in Scotland*, W. Green Ltd, Edinburgh, 1964, p192.

²⁵ The writer is of the belief that in many modern day cases this situation still prevails.

²⁶ The English criminal cases of poisoning were noted within the six groups of poison in this study- See Watson, K., *Poisoned Lives: English Poisoners and their Victims*, Hambleton and London, London, 2004, p33.

stands out strongly from that which existed one hundred years ago and of course, the contrast to the accuracy of diagnosis in the early nineteenth century is even more remarkable.

Thus, in the following chapters, accounts and analysis of Scottish poisoning trials are tied to examination of the law and development of medico-legal science to advance the following arguments.

Firstly, the thesis uses the case study evidence to propose the basic conclusion that murder and attempted murder by poison was not rare in nineteenth century Scotland. In contrast to the foci of scholarship concerning the headline cases of poisoning, the subsequent chapters demonstrate simple access to a variety of deadly poisons and abundant motives for their use by the disadvantaged of society.

Secondly, several socio-economic points provide reasoning for the fact that many of the cases of the crime of poisoning investigated were committed by disadvantaged individuals. Poisons were cheaply available to even the poorest and were sold in a largely unrestricted fashion. Further, given the crushing poverty and substantial social inequality of the period, many turned to poison in desperation or for financial gain. Finally, given the prevalence of fatal diseases and infant mortality amongst the rural and urban poor, potentially suspicious deaths could be expected to pass unnoticed.

Thirdly and most importantly, the analysis of the poisoning cases in Scotland sheds light on development of the medico-legal profession, and this latter also provides crucial weight to the argument that murder by poisoning must have been much more common than the court records would suggest. Many of the cases considered in the following chapters saw a great deal of evidence mounted against the accused but juries passed Not Proven verdicts. The technical limits of the new science of forensic toxicology were stretched during this period. Moreover, the trust which the public and the law were willing to extend to forensic medicine was stretched also. The variability of the quality of forensic work and the trust placed in medical witnesses is shown to be a hallmark of Victorian justice in the case of detecting and proving the subtle arts of poisoning. The prevalence of lenient sentences and Not Proven verdicts attests to unreliability in science and distrust of the concreteness of medical evidence by juries.

Such facts corroborate the argument that many cases would never have been detected by unskilled practitioners, and thus, the records of the Court give an unrealistically low figure for the true extent of poisoning.

Today we are used to scientific analysis and legal or criminal investigation working hand-in-hand in pursuit of truth and justice. The study of poisoning enables a documentation of the gradual, halting alliance between the law and science in the Victorian period as the legal establishment caught up to the potential scale of the problem of easy and previously undetectable murder occasioned by the desperation of the circumstances of life of the socially disadvantaged.

Chapter 2

Arsenic, A Poison For Rats Or An Excuse For Murder

2.1. Introduction

Arsenic is a metallic element, which exists in the earth's crust and has been recognised as a poison since pre-Christian times.¹ Further, it was the main poison used in the extraordinary “*Affair of the Poisons*”, which was one of the most notorious incidents of the reign of Louis XIV in France.² It has been described as The Queen of Poisons and Sir Robert Christison, the eminent nineteenth century Scottish toxicologist and physician, stated that “(A)rsenic is the poison most frequently chosen for the act of committing murder.”³ Arsenic had been a preferred method of poisoning in the Middle Ages and in the Renaissance – the infamy of the Borgias for removing

¹ Polson, C.J., and Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London, 1981, p181.

² Louis XIV, 1654 - 1712. The Poison Affair was a murder scandal in France during the reign of King Louis XIV. It launched a period of hysterical pursuit of murder suspects, during which a number of prominent people and members of the aristocracy were implicated for poisoning and witchcraft. The poison of choice was usually arsenic, and matters rapidly developed into a *cause célèbre*, with unsavoury alchemists and fortune-tellers denouncing each other, and naming more and more of the aristocracy as their clients. Before it was over, three hundred people had been tortured, hanged or burned at the stake, and scandal had touched some of the highest in the land. See Mossiker, F., *The Affair of the Poisons*, Sphere Books Ltd, London, 1975.

³ See Bartrip, P., *Medical History*, vol 36, 1992, p54, The Arsenic Act, 1851 and the Prevention of Secret Poisoning. Sir Robert Christison (1797-1882) received his M.D. degree from the University of Edinburgh in 1819 and later returned there as a Professor of Materia Medica after working as a house physician and studying under M.J.B. Orfila, the distinguished toxicologist. His treatise on poisons (1829) was recognised as a classic and several editions were published.

their rivals and enemies has often been associated with their use of arsenic⁴

This history aside, throughout the early 19th century many reports appeared in prominent medical journals promoting the use of arsenic as a remedy.⁵ This was in relation to common white arsenic - a crystalline white cake also known as arsenious acid. Indeed, one medical paper even suggested that arsenic was “entitled to hold a situation of eminence” with relation to its supposedly beneficial properties⁶. Contrary to these optimistic new attempts to develop arsenic as a cure for ailments, as early as 1809 reports began to appear in the Edinburgh Medical Journal, founded in 1805, in relation to arsenic being used as a means to commit suicide.⁷ The journal contained articles of interest to members of the medical profession and such reports were soon

⁴ Mass homicide using arsenic is said to have been practised by the Borgias, an Italian noble family of Valencian origin in the 15th and 16th centuries. The patriarch of the family, Rodrigo Borgio, became a bishop, cardinal, vice-chancellor of the church and eventually Pope. Other members of the Borgia family were Lucrezia Borgia and Cesare Borgia, daughter and son of Rodrigo Borgia, respectively. Both Cesare Borgia and his sister Lucrezia frequently employed the use of a white powder they referred to as *La Cantarella* and which was almost certainly arsenic trioxide- Emsley, J., *The Elements of Murder: a History of Poisons*, Oxford University Press, Oxford, 2005, pp141-142. In the 17th century a poisonous liquid was much used by young wives in Italy who wanted to get rid of their husbands. This liquid was named “Aqua Toffana” after the woman called Toffana who had invented the recipe and it was sold in phials, which bore the representation of a saint, usually Saint Nicholas of Bari. Toffana managed to sell “Aqua Toffana” under the pretence that it helped a woman’s complexion and it has been estimated that Toffana aided the murder of over 600 people, usually husbands- Polson, C.J., and Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London,1981, p181.

⁵ Medical cases in which the use of arsenic was indicated included rheumatism, vermes, dyspepsia, hypochondria, epilepsy, hysteria, convulsions in children, palpitations and headaches. See Hill, G.N., *EMJ*, vol 5, 1809, pp19-27, The Medicinal Effects of Arsenic, Externally and Internally Applied; Jenkinson, J., *EMJ*, vol 5, 1809, pp309-319, On the Cases in Which the Use of Arsenic is Indicated.

⁶ Hill, G.N., *EMJ*, vol 5, 1809, p20, The Medicinal Effects of Arsenic, Externally and Internally Applied.

⁷ Yelloly, J., *EMJ*, vol 5, 1809, pp389-393, A Case of Death Produced by Arsenic; Cook, E., *EMJ*, vol v, 1810, pp200-205, Three Cases of Suicide with Arsenic; Ward, H., *EMJ*, vol 33, 1830, pp61-65, A Case of Poisoning with Arsenic.

followed by accounts of trials for poisoning with arsenic.⁸ Given the ease of procuring arsenic in this period, however, the existence of such reporting is predictable.

In the early nineteenth century even the most deadly poisons were indiscriminately sold. In particular, arsenic was readily obtainable from druggists, doctors, chandlers, oilmen and village shopkeepers, and found in many patent medicines.⁹ There were no legislative controls over the sale of arsenic in the early nineteenth century and any person, however uneducated and ignorant, was able to sell medicines and poisons such as arsenic.

Arsenic appears to have been the most popular substance used as a poison due to the ease with which it could be obtained and due to the high level of recognition it enjoyed as a result of its historical infamy. It was sold openly to control vermin in the form of rat poison and also used extensively in the glassmaking and dying industries. Further, following the discovery of copper arsenite (a green pigment), arsenic was used extensively for the printing of wallpapers, to colour wax candles, in the dyeing of cotton and linen and even to paint some children's toys.¹⁰ It was also used as food dye for sweets such as green blancmange. Such widespread utility was increased by its

⁸ Christison, R., *EMJ*, vol 27, 1827, pp 441-472, Account of the Medical Evidence in the Case of Mrs Smith, Tried at Edinburgh in February Last for Murder by Poison; Cowan, S., *EMJ*, vol 1, 1840, pp 651-661, The Trial of Marie Lafarge; Christison, R., *EMJ*, vol 1, 1855-1856, pp 625-761, Account of a Late Remarkable Trial for Poisoning with Arsenic; Hobson, B., *EMJ*, vol 5, 1859, pp81-82, Poisoning with Arsenic.

⁹ Such patent medicines included Fowlers solution initially for use in fevers, neuralgia, syphilis, epilepsy, skin disorders and then later as a tonic. It was composed of arsenic trioxide, potassium bicarbonate, hydroxide or carbonate and water and often causes skin cancers. Indeed, until relatively recently people born in the late 1800s/early 1900s were found by their doctors to be suffering from Bowen's disease, a form of skin cancer— caused by this tonic. See Emsley, J., *The Elements of Murder*, Oxford University Press, 2005, pp104-106.

¹⁰ Copper arsenite was first made in 1775 by the chemist Karle Scheele (1742-1786); because it was a beautiful green colour. Scheele realised that he could make money from the manufacture of it as a new pigment. See Emsley, J. *The Elements of Murder: A History of Poison*, Oxford University Press, Oxford, 2005, pp117-118.

cheapness and it was affordable by even the lowest classes.¹¹ From the results of the attention arsenic received in scientific journals, as well as reports in the national press, there arose an increased knowledge of arsenic among the populace.¹²

2.2 Typicality of Arsenic Poisoning Cases and Diagnostic Procedures

Between 1800 and 1913 information relating to thirty-two trials for murder and attempted murder with arsenic in Scotland were discovered in the course of this research. In a further case the accused was charged with culpable homicide.¹³ A final case was terminated when the accused, on being questioned by a police-sergeant on suspicion of arsenic poisoning, ran to his bedroom and swallowed the contents of a phial of prussic acid resulting in instant death.¹⁴ Nineteen (59%) of the murder trials involved women and thirteen involved (41%) men.¹⁵ Indeed, of the total of eighteen executions for murder by poison in Scotland between 1800 and 1913, eleven (61%)

¹¹ Due to its cheapness and ease of availability obtaining arsenic would have been no more difficult than purchasing aspirin today – costing one penny to two pennies an ounce, during the 19th century: Pierce, M., *London Medical Review and Magazine*, vol 3, no. X1V, 1800, pp 106-108, Current Prices of Drugs in the London Market; Pierce, M., *London Medical Review and Magazine*, vol 4, no. X11, 1884, Current Prices of Drugs in the London Market. Arsenic was, therefore, accessible to all classes of persons in pre-Victorian and Victorian society providing a simple and inexpensive recipe for various requirements.

¹² From this the writer believes that many varied means were resorted to for the purposes of administering arsenic as a poison. Of note is that often the target for an arsenic prescription in the Victorian era was sexual potency. *Arsenicon*, from the Greek, means potent. By 1882 the habit was being reported in provincial newspapers: “We are perfectly aware that men-about-town are as much in the habit of taking these dangerous drugs, strychnine and arsenic and what-not, as they are of drinking champagne and smoking tobacco.” – Foucault, M., *The Birth of the Clinic: An Archaeology of Medical Perception*, Routledge, London, 1963, p156.

¹³ Case of Andrew Paterson.

¹⁴ Case of John Hutchison.

¹⁵ See Appendix 1. Note also that one of the trials was a double trial- that of Sarah Fraser and John Fraser in 1852 for the murder of their father- See Appendix 1.

were for poisoning with arsenic.¹⁶ Of these eleven executions, eight (73%) were women and three (27%) men.

It would appear that arsenic was the most popular poison for committing murder in the past in Scotland and thus to begin to build the case for the scholarly and legal underestimation of homicidal poisoning, arsenic must be the first point of departure.. More than half of all my poisoning cases 54% involved the use of arsenic. This concurs with English figures and as Watson has stated, “(T)he story of poisoning in England and Wales is in many ways a chronicle of the rise and fall of arsenic.”¹⁷ By contrast, only six strychnine poisoning cases, one prussic acid case, twelve opium cases, one phosphorous case and seven cases for poisoning with corrosive acids were discovered in this research. Further, my figures would suggest that arsenic was the poison favoured by women.

¹⁶ The figure of 20 is from my own research in relation to murder and attempted murder by Poison in Scotland and relates to the number of executions that I have come across. The writer is fully aware that the figure of 20 may be wrong due to records not having been kept in the past, material relating to crime from the past having been destroyed and no statistics for this type of crime having been kept. To be noted is that the figure of 20 includes the execution of John McMillan in 1810 for the murder of Barbara McKinnel with muriate of mercury- See Young, A.F., *The Encyclopaedia of Scottish Executions*, Eric Dobby Publishing, Ltd, Kent, pp70-71. Also the execution of Dr Pritchard in Glasgow in 1856 for the murder of his wife and mother-in-law with tartarised antimony and aconite and opium - See Roughead, W., *Trial of Dr Pritchard Notable Scottish Trials*, W.Green & Sons Ltd, W.Hodge Co. Ltd, London, 1912. These executions are included in an attempt to provide figures, which are as accurate as possible, although these cases are not discussed in this thesis.

¹⁷ Between 1759 and 1914 there were 237 cases in England for poisoning with arsenic. This figure represents 66% of all poisoning cases in England within the group of six poisons in this research for that time period- See Watson, K., *Poisoned Lives: English Poisoners and their Victims*, Hambleton and London, London, 2004, p33. Further during the time period 1739-1878 there were 19 trials for arsenic poisoning at the Old Bailey which represents 40% of all poisoning trials at the Old Bailey within the field of the chosen poisons for this research- See Forbes, T.R., *Surgeons at the Old Bailey*, Yale University Press, London, 1985, Table 8.

Crowther and White (among other writers) have suggested that homicidal poisoning was rare in the past.¹⁸ The research in this study suggests otherwise and this is particularly apparent in the case of homicidal poisoning with arsenic where, even considering the figures above, it is highly likely, as will be argued in later sections, that the true number of murders by arsenic poisoning are severely masked by failings in diagnosis and in the inability to tell murder from accidental poisoning.¹⁹

The sheer ubiquity of arsenic in the Victorian market and household hugely increased the potential for the concealment of murder. In most of the Scottish trials for murder and attempted murder with arsenic there are the invocations of the “rat excuse” by the accused.²⁰ Nevertheless, the vehicle of choice for administration was in most cases porridge or tea, and was the exemplar of simplicity.²¹ In all cases studied the victim’s symptoms were typical of those produced by arsenic. These symptoms included vomiting, purging, and burning pain, excessive thirst and painful cramps in the stomach and bowel. However, to the Victorian medical establishment, other causes, such as food poisoning and cholera, might easily have produced these same symptoms.²² Little or no dependence could be placed in the past on being able to prove arsenic poisoning by symptoms and post-mortem appearances alone.²³

¹⁸ Crowther, A and White, B., *On Soul and Conscience: The Medical Expert and Crime: 150 Years of Forensic Medicine In Glasgow*, Aberdeen University Press, 1988 p19; Smith, F., *Cause of Death*, Orbis Publishing, London, 1980, p201; Advocate, H.M. v. Elder (or Smith) (1827) Syme 71, p213.

¹⁹ See Conclusion.

²⁰ The claim by the accused being that they possessed white arsenic for the purpose of poisoning hypothetical rats.

²¹ Since the compound of arsenic is very soluble in hot water and indeed almost tasteless and colourless, it could easily be administered without arousing suspicion. Porridge and gruel are said to be excellent vehicles being able to hold massive quantities without suspicion.

²² Between 1831-1832, cholera struck for the first time in Scotland killing about 10,000 people, and returned with almost equal severity in 1848-1849. The third outbreak in 1853-1854 was less lethal and the final in 1866-1867 made little impact on the death rates- Fraser, W.H., & Morris, R.J., *People and Society in Scotland 1830-1914*, Arrow Smith Ltd, 1995, p24.

²³ See Orfila, M.J.B., *Annales D’Hygiène et de Médecine Légale*, Mémoire sur L’empoisonnement par L’acide Arsénieux, 1839, pp36-361- “The physician who, judging from the symptoms and morbid appearances only, should affirm that a person had been poisoned, would be blameable. He would still be more culpable if he were to depone to this effect without having actually detected poison.”

Indeed, the courts' decisions in poisoning trials in the nineteenth century appeared to depend solely on the results of chemical analysis. Until 1836, when Marsh's test for arsenic was first introduced, the means of detecting arsenic was both presumptive and inadequate.²⁴ In fact the earliest test for arsenic to receive universal recognition was the odour of garlic produced when suspected food or stomach contents were thrown on to red hot coals.²⁵ The continuing inexactness of arsenic testing is the most important area in which the halting relationship between forensics and the law was played out. Science seemed to promise great breakthroughs in detection, but in many cases delivered inconclusive results and could not meet the conditions of proof which nineteenth century juries expected.

Of the fifteen trials discovered before 1836, nine of these resulted in 'Not Proven' or 'Not Guilty' verdicts (60%). Given this inability to reach a definite conclusion in many cases, there must therefore be reasonable doubt as to how many wrongful verdicts were given due to juries reaching decisions by wholly trusting the opinions of respected (though flawed) medical witnesses. Also worthy of consideration is the fact that many medical men in rural areas had never knowingly seen cases of arsenic poisoning, nor carried out experiments in arsenic testing.²⁶

Further doubt on the correctness of verdicts in poisoning trials must surely exist even after the introduction of Marsh's test in 1836. This is because the test, despite

²⁴ In 1836 James Marsh, employed as a chemist at the Royal Arsenal in Woolwich and as an assistant to Michael Farrady at the Woolwich Military Academy, described his new reduction test for arsenic. With it he was able to separate very minute quantities from gruel, soup, porter and other alimentary liquors. He could, he said, detect as little as 1/120 of a grain (0.5mgm) of an arsenic compound, and reported that his test was unambiguous providing that precautions were taken so that the zinc used in the reaction was not in itself contaminated with arsenic- See Forbes, T.R., *Surgeons at the Old Bailey*, Yale University Press, London, 1985, p136. See also Appendix 2 for other historical tests employed for detecting arsenic.

²⁵ See Campbell, W.A., *Chemistry in Britain*, vol 1, 1965, p198 Some Landmarks in the History of Arsenic Testing.

²⁶ Information as to the effects of the tests for arsenic was in many cases drawn solely from books. See in particular trial of Mary Elder or Smith – Advocate. H.M. v. Elder (or Smith) (1827) Syme 71, p65. See also case of James M'Kerle where a doctor on initially examining the victim, John Stewart, pronounced him dead, even although he was not – Advocate. H.M. v. M'kerle (1842) Broun 429, p432.

furnishing good collateral evidence, was complicated and unfit to be entrusted to those unaccustomed to careful chemical manipulation.²⁷ The Marsh test was, therefore, anything but foolproof.²⁸ Hugo Reinsch's test for arsenic published in 1841, although requiring a much lesser degree of skill also met with much criticism for its lack of reliability.²⁹ Courts became reluctant to admit chemical evidence, and as late as 1889 the judge presiding at the poisoning trial of Mrs Maybrick in England remarked, "One has to take a great deal of scum from the testimony of skilled witnesses."³⁰

Serious problems thus existed in trials for murder and attempted murder by arsenic, as evidence, even that of scientific authorities, amounted often to nothing more than the circumstantial. Indeed, the first question in a murder trial was did the person die of poison³¹ or natural disease. Upon that question was required medical evidence both clear and conclusive.³² Given the many uncertainties and errors in medical science during the nineteenth century many juries, and indeed, much popular sentiment distrusted this branch of the law.

²⁷ See Campbell, W.A., *Chemistry in Britain*, vol 1, 1965, p201, Some Landmarks in the History of Arsenic Testing. See also Gamjee, A., *EMJ*, vol 10, 1864-1865, p408, On an Alleged Fallacy in Marsh's Process for the Detection of Arsenic

²⁸ See the 1840 trial of Marie Lafarge in France in which contradictory results were obtained- Saunders, E., *The Mystery of Marie Lafarge*, Praeger, London, 1931.

²⁹ See English trial of Thomas A. Smethurst in 1859 where unsatisfactory results were yielded from this test- Parry, L. A., *The Trial of Dr Smethurst*, Notable British Trials, W. Hodge & Co, Edinburgh & London, 1931, pp85-101.

³⁰ Campbell, W.A., *Medical History*, vol 25, 1981, p202, The History of the Chemical Detection of Poisons.

³¹ In particular see trial of Mary Elder or Smith in 1827 – Advocate. H.M. v. Elder (or Smith) 1827 Syme 71, p128.

³² For chemical tests used to determine the presence of arsenic see Appendix 2. For post-mortem appearances of a person who has died from the effects of arsenic poisoning see Appendix 3.

2.3 Case Histories: An Overview

The majority of the recorded cases for poisoning with arsenic in Scotland occurred before mid-century.³³ Setting this fact in a broader historical context, it is evident that the decrease may be attributed to the passing of the Arsenic Act in 1851.³⁴ Prior to this Act there were no restrictions on the sale of poisons in the UK.³⁵ Indeed the mid-Victorian era appears to have been of pivotal significance in the history of law relating to poisoning.³⁶ Following the passing of the Arsenic Act legislation concerning other poisons was to quickly follow.³⁷ The Arsenic Act attempted to

³³ 24 out of the 34 cases – 71%.

³⁴ Throughout the 1840s many proposals were made for restricting arsenic sales or for colouring it to prevent mistakes. See for e.g.: Holloway, D., *EMJ*, vol 53, 1840, p252, Sale of Arsenic, where it was suggested that arsenic used in medicine should be mixed with sulphate of iron and the cyanide of potassium which would give the arsenic a peculiar colour when mixed in solids or fluids. See also Parliamentary Papers, 1841, VII: *Third Annual Report of the Register General*; in which the chemist Farr suggested that suicide by poisoning could be reduced if medicines containing poisons were available only on prescription, if arsenic was no longer supplied for vermin control, and poisons used in industry obtainable only by those holding an employer's certificate.

³⁵ There was, however, legislation to recognise the crime of poisoning. See James II, 7th Parl. ch. 31 & ch. 32, 1450, forbidding the import of poisons. Also 6 Geo. IV, ch. 126, 1825, in which it was enacted that it was a capital crime to murder another person with poison.

³⁶ The passing of the Arsenic Act in 1851 seems to have been mainly as a result of pressure on the government from the Provincial Medical and Surgical Association and the Pharmaceutical Society of Great Britain. This is in combination with a figure of 239 for the number of trials in the UK for murder and attempted murder by poison in the years 1839 to 1849 and growing public concern over arsenic. This seems to have been sufficient to ensure the passage of the Bill, introduced by Lord Carlisle, Through Parliament. See Parliamentary Papers 1850, XLV: *Returns of the Number of Persons Tried in the UK for Murder and Attempts to Murder by the Administration of Poison. In Each Year 1839-1849 Inclusive*, pp447-453.

³⁷ The Arsenic Act of 1851 did not restrict in any way those who could sell arsenic and although the Pharmacy Act of 1852 gave the Pharmaceutical Society the power to hold exams and issue professional certificates anyone could still call himself a chemist or druggist. Indeed, it was not until the passing of The Pharmacy and Poisons Act of 1868 that the selling of poisons was restricted to doctors, pharmacists and druggists who were registered. See Pharmacy Act 1852; Pharmacy Act 1868; Pharmacy Acts Amendment Act 1898; Poison and Pharmacy Act 1908.

impose strict controls on sale and purchase of pure arsenic.³⁸ Despite this, however, sales in arsenic, and in particular uncoloured arsenic, continued.³⁹ Legislation did not have the fully desired effect and control of the trade in arsenic was little helped by the frequent arousal of the public's interest and fascination during the mid nineteenth century in this metallic poison. Interest was stoked as a consequence of the many newspaper and journal articles concerning both the 'arsenic eaters' of Styria and the

³⁸ The regulations imposed by the Act were that details of every sale of pure arsenic were to be entered in a Poison Book and that arsenic could only be sold to a person known to the seller unless in the presence of a witness known to the seller. Further, arsenic could not be sold to anyone under the age of twenty one. In addition, pure arsenic had to be coloured with soot or indigo unless the buyer stated that this would render it useless for his purposes. For any bulk sale of ten pound or more, which was common among farmers, manufacturers and wholesalers the arsenic was not required to be coloured. The Act therefore did not apply to arsenic to be used for commercial purposes or medicines containing arsenic. It is interesting to note that originally it was proposed that arsenic be sold only to men. This proposition was, however, dropped. See Bartrip, P., *Medical History*, vol 32, 1992, p65, A Pennurth of Arsenic for rat Poison: The Arsenic Act 1851 and the Prevention of Secret Poisoning.

³⁹ From the number of deaths still taking place from uncoloured arsenic (white arsenic) it was clear that vendors were not adhering to the law. See Parliamentary Papers, 1864, XXVIII: *Sixth Report of the Medical Officer to the Privy Council*, Appendix 16, p759.

benefits of using arsenic as a cosmetic or in medicine.⁴⁰

Trials for murder and attempted murder with arsenic in Scotland, thus, continued after 1851 although in reduced numbers.⁴¹ The majority of these later trials involved women.⁴² Among these later trials is that of Madeleine Smith in 1857⁴³, considered to

⁴⁰ See Heisch, C., *EMJ*, vol 5, 1858-1860, p1137, The Arsenic Eaters of Styria; MacLagan, D., *EMJ*, vol 10, 1864-1865, p200, On the Arsenic Eaters of Styria –“ On the hillsides of the Tyrol, in Hartburg, Styria and other districts, local stablemen perfected a technique that was passed on from father to son. This began with the eating of a millet-sized piece of white arsenic which progressed over the years to a piece the size of a pea. This all started, apparently with horses, and the fact that a little arsenic given daily in the corn improves the coat. In fact unscrupulous horse-handlers the world over have used arsenic as a “coat shiner” in lieu of elbow grease, despite the threat of prosecution under Poisons legislation. A little bit of what was considered good for horses was, therefore, deemed appropriate for human use and these mountain men were observed by the medical professors who examined them, to be robust, vigorous and long lived. They were known as Arsenophagists and by the middle of the nineteenth century many men in the UK and America had acquired the arsenic habit. It is interesting also to note that many people supposed the origin of the vampire came from the virtually unchanged dead bodies of arsenic eaters in Styria when these bodies were dug up from graveyards about every twelve years to allow room for new burials. This is because it had long been believed that arsenic preserved the body tissues and that the body is well preserved even when exhumed long after burial. With regards to the use of arsenic as a cosmetic the use of white arsenic to cosmetically whiten skin is a practice said to be one of the vanities of the nineteenth and early twentieth centuries, but which almost certainly dates back to Elizabeth I’s reign and beyond - See Wilson, A.N., *The Victorians*, Hutchinson, London, 2002, p550. With reference to arsenic in medicine, Fowler’s solution which contained potassium arsenite with a little lavender water was reputed to cure all sorts of ailments ranging from neuralgia to syphilis and epilepsy. Indeed, no less than James Begbie, Vice-President of the Royal College of Physicians of Edinburgh and Queen Victoria’s doctor, when she was in Scotland, endorsed this product- See Emslie, J., *The Elements of Murder*, Oxford University Press, 2005, pp105-106.

⁴¹ Out of the 34 trials between 1800 and 1913 in Scotland for arsenic poisoning 7 of these were after the passing of the Arsenic Act in 1851 – 21%.

⁴² 4 out of the 7 trials involved women – 57%.

⁴³ Countless books, articles and plays have been written about this trial and even a motion picture “Madeleine” told the story in 1949. See for eg: Tennyson, J.F., *Trial of Madeleine Smith*, Notable British Trials, W. Hodge & co. Ltd, Edinburg & London, 1927; Browne, G.L., & Stewart, C.G., *Reports of Trials for Murder by Poisoning*, Steven & Sons, London, 1883, pp294-236; MacCowan, D., *Murder in Victorian Scotland*, Praeger Publishers, London, 199, p321-353; Crowther, A., *Scottish Local History*, vol 29, 1993, pp23-27, Crime and Punishment.

be one of the greatest criminal trials for poisoning with arsenic and one of the most sensational trials of the nineteenth century. However, of equally significant medico-legal importance are the other, lesser publicised arsenic poisoning trials here described. Without giving consideration to these other trials it is impossible to portray a clear and concise history of arsenic poisoning in Scotland. It has been boasted by the journalist Jack House that all the best Scottish murders took place in affluent areas of Scotland.⁴⁴ This implies that only respectability provides the characteristics for a poisoning trial to be of medico-legal importance, significance, and of course historical interest. Of the thirty-four poisoning trials covered herein only one of the accused came from what could be described as a “position of respectability”.⁴⁵ It is only in grasping the totality of the phenomenon of arsenic poisoning, irrespective of class concerns or the showiness of individual trials, that full evaluation of the patterns of motive and prosecution of crime can be laid out.

2.4 Earliest Records: 1800 - 1826

Three arsenic poisoning cases from the period before 1816 form part of this study. In 1806 Margaret Cunningham poisoned her husband by putting arsenic in his porridge, Helen Kennedy who poisoned her master Thomas Stohard using the same method in 1811, and finally Christain Sinclair poisoned her niece with arsenic laced porridge in 1813. In all cases the victims died and both Margaret Cunningham and Christain Sinclair were found guilty and executed.⁴⁶ A ‘Not Proven’ verdict was returned in the case of Helen Kennedy.⁴⁷ Little detailed information exists on these three cases, but noteworthy is the use of porridge in each case for the administration of arsenic.

The aforementioned ease with which any person could obtain arsenic, particularly before 1851, is illustrated by the fourth case, that of thirteen year old Catherine

⁴⁴ See House, J., *Square Mile of Murder*, Malinder Press, London, 1975, p3. In particular see the trials of Madeleine Smith in 1857, Jessie McLachlan in 1862 and Dr. Pritchard in 1865.

⁴⁵ See trial of Madeleine Smith in 1857.

⁴⁶ See Young, A.F., *The Encyclopaedia of Scottish Executions*, Eric Dobby Publishing Ltd, Kent, p69 & p74 for source of information regarding Margaret Cunningham and Christain Sinclair.

⁴⁷ Record of the Lord Advocate AD14/12/66.

Clerk.⁴⁸ At Edinburgh, in July 1816, Catherine Clerk, a domestic servant, was charged with attempting to murder her mistress, Christian Ritchie. Arsenic was alleged to have been dropped into a teapot by Catherine from which Mrs Ritchie drank a cup of tea and then quickly developed the symptoms of arsenic poisoning, although she later recovered.

Following analysis, evidence was presented to show that a paper packet, which had been removed from the teapot, contained sulphate of arsenic.⁴⁹ A statement from druggist Andrew Craigie, confirming that Catherine had purchased a penny worth of King's Yellow on Friday 24th May for killing flies went further to establishing her guilt.⁵⁰ Indeed, Catherine's own declaration in which she stated that she had both purchased the arsenic and placed it in the teapot made it conclusive that Catherine had perpetrated the crime of which she was accused. Accordingly she was found guilty, despite there being little evidence of real motive.⁵¹

The fifth case, that of Robert Dingwall who poisoned his wife in 1818 with arsenic and was found not guilty, possesses no useful extant details.⁵² The sixth case is that of Anne McEachern of Ardehiaveg, Argyle who, on the 17th of April 1820 put arsenic into a bowl of porridge which she then gave to her husband, Neil McKinnon. Neil survived the incident and Anne was found guilty of attempting to murder him.⁵³ The seventh case concerns George Thom from Aberdeen who poisoned his wife and her two brothers with arsenic with a view to gaining an inheritance that they had been left. Whilst all suffered severe symptoms of arsenic poisoning only William Mitchell died

⁴⁸ Record of the Lord Advocate AD14/16/23.

⁴⁹ Record of the Lord Advocate AD14/16/23 per Dr. Duncan of Edinburgh.

⁵⁰ King's Yellow is a tri-sulphide of arsenic (arsenic combined with sulphur) - AS₂S₃, also known as orpiment or yellow arsenic. It owes its poisonous properties to the presence of arsenious acid and was used as a pigment, for killing flies and as a depilatory. It was much used in India and other parts of the East both as a medicine and as a poison. Further, this King's Yellow compound was used by illustrators and decorators.

⁵¹ For the commission of this crime Catherine was banished from Scotland for 10 years.

⁵² See Alison, A., *Principles of the Criminal Law of Scotland*, Blackwood, Edinburgh, p166 and Appendix 1.

⁵³ See Record of the Lord Advocate AD14/21/195 and Appendix 1.

and George Thom was hung on November 6th 1821 for his crime.⁵⁴ The eighth case occurred in Glasgow, and is that of Helen Rennie who, in 1822, was charged with administering poison to her son. Helen's son died as a result, but at trial she was found not guilty.⁵⁵ No further details to explain this verdict are extant.

2.5. Three Doses: The Cases of 1827

The criminal records of 1827 are notable for the occurrence of three arsenic poisoning trials, of which two resulted in 'Not Proven' verdicts.⁵⁶ The first 'Not Proven' verdict arose in the ninth case of this enquiry that of Mary Elder or Smith, the wife of a farmer at Denside, near Dundee. At this farm a servant, Margaret Warden, became pregnant by Mrs Smith's youngest son. With a view to procuring a miscarriage and averting a scandal, Mrs Smith frequently gave Margaret doses of "medicine" that had the appearance of cream of tartar. As a consequence Margaret became seriously ill with all the symptoms of arsenic poisoning.

A certain Dr. Taylor, on examining Margaret, formed the impression that she had cholera.⁵⁷ However, Margaret expressed both to her mother and to a fellow servant her suspicions of foul play on the part of her mistress just before dying: "ye ken wha is the occasion o' me lying here. My mistress gave me"⁵⁸ Following the burial of Margaret, suspicion of murder was rife and her body was promptly exhumed and examined by three surgeons under direction of the Procurator Fiscal.

⁵⁴ See Young, A.F., *Encyclopaedia of Scottish Executions 1750 to 1963*, Eric Dobby Publishing, Kent, 1998, p91 and Appendix 1.

⁵⁵ Carse, William, "Trial and Sentence of all the Prisoners who have Appeared Before the Court of Justiciary Since its Opening on Thursday the 18th April 1822", Broadside from Archives of the National Library of Scotland.

⁵⁶ See Appendix 1: Trial of Mary Elder or Smith – Advocate, H.M. v. Elder (or Smith) (1827) Syme 71; Trial of Margaret Wishart – Record of the Lord Advocate AD/14/27/179; Trial of John Lovie – Advocate, H.M. v. Lovie (1827) Syme's Justiciary Reports, Appendix no. 2, p99.

⁵⁷ The disease was prevalent in the country at that time and the symptoms were all such as cholera produces.

⁵⁸ Advocate, H.M. v. Elder (or Smith) (1827) Syme 71, p99.

Post-mortem examination pointed to arsenic poisoning and seven different liquid tests, a reduction test and the sulphuretted hydrogen test were applied to the contents of the stomach and intestines.⁵⁹ The information for these tests was drawn solely from books, as these were the investigating surgeon's first experiments with arsenic. Despite this, however, the surgeons were convinced that they had detected arsenic and Mrs Smith was apprehended on a charge of murder. The experiments and testimony of Dr. Christison backed up this conviction during the trial.⁶⁰ Thus, considering the opinions and observations of four medical men there appeared to be clear and conclusive evidence that death in this case had been caused by poison.

Direct testimony as to how a poison was administered is highly unlikely in any such case.⁶¹ Thus circumstances on which reliance can be placed for establishing guilt or innocence must be examined. The first important circumstance is the possession of the poison by the accused. Dr. Dick of Dundee stated during the trial that he had sold Mrs Smith about an ounce and a half of arsenic to poison rats.⁶² The prisoner was, therefore, proved to have had in her possession poison, and poison of the particular kind found in the body of Margaret Warden.⁶³ In this case the accused had every opportunity for administering poison secretly in the many "Medicinal" drinks she gave Margaret in an attempt to cause a miscarriage. There being thus acquisition of the poison and opportunities for secretly administering it, the presence of motive could be sought. From the trial evidence it is obvious that Mrs Smith wanted to cause the abortion of Margaret's pregnancy, and, in either in the course of this attempt, or in despair of accomplishing her purpose, perpetrated the crime of murder.

⁵⁹ Appendix 3, appearances -: 1, 2, 3, 13. Appendix 2, tests -: A9, A14, B3, B4, B5, B6, B7, B10, B14.

⁶⁰ Christison, R., *EMJ*, vol 27, 1827, p441, Account of the Medical Evidence in the Case of Mrs Smith, Tried at Edinburgh in February Last for Murder by Poison.

⁶¹ The case of Nairn and Ogilvy (Scotland, August 1765) is probably the only one in which the accused was seen to mix the poison in the food, which was afterwards administered – Burton, J.H., *Criminal Trials in Scotland*, Chapman & Hall, London, 1852, p25. Although see also the case of Lydia Dodds in the chapter on poisoning with opium in, which Lydia was seen to put a cup to her child's mouth and pour in a brown liquid – Record of the Lord Advocate AD14/68/131.

⁶² Advocate, H.M. v. Elder (or Smith) (1827) Syme Appendix 1, p111.

⁶³ Despite Mrs Smith being proved to have been in the possession of arsenic, she initially vehemently denied this – Ibid p111.

An attempt by the defence to show that Margaret had committed suicide broke down. However, despite the many circumstances seeming to prove deliberate poisoning, a trial of twenty-two hours resulted in a verdict of ‘Not Proven’ being returned by the jury.⁶⁴ Such a verdict seems, to modern reckoning, unlikely and indeed Lord Cockburn, who defended Mrs Smith, admits his client’s guilt in his book *Circuit Journeys*.⁶⁵ The panel was, thus, allowed to go free perhaps to poison again.⁶⁶

The second case in 1827 was that of John Lovie. Once more, it seems unlikely that justice was served. The setting for murder was a farm and, as common, the excuse of rats was employed. John Lovie, a farmer, near Fraserburgh had seduced one of his maids, Margaret McKessor. Margaret fell pregnant and mistakenly believed Lovie would marry her. At the end of July 1827 Lovie enquired of his ploughman, Alexander Rannie, if he knew of substances which would produce an abortion and as to the effects of jalap, laudanum and arsenic in this respect.⁶⁷ Following this Lovie called at a chemist’s shop in Fraserburgh where he purchased both jalap and an ounce of arsenic.⁶⁸ The latter was bought under the pretext that, at the farm, “they were much infested with rats.”⁶⁹

⁶⁴ A Scottish jury is not restricted to the two verdicts of Guilty and Not Guilty. There is a third possibility, a verdict of Not Proven. After such a verdict the accused is acquitted and cannot be tried on the same charge. In this respect it has the same effect as a verdict of Not Guilty. The Not Proven verdict is often known in Scotland as the “bastard verdict” and over the years has faced considerable criticism – see Conclusion.

⁶⁵ Cockburn, H., *Circuit Journeys*, David Douglas, Edinburgh, 1888, p12- “Lockhart mentions Scott as having gone to see my old client Mrs Smith, who was guilty, but acquitted of murder by poison.”

⁶⁶ See Scott, W., *The Journal of Sir Walter Scott*, p12; “Well Sirs! All I can say is that if that woman was my wife I should take good care to be my own cook.”

⁶⁷ Advocate, H.M. v. Lovie (1827) Syme’s Justiciary Reports, Appendix no. 2, p26.

⁶⁸ Jalap is the tuber of *Ipomoea Jalapa*, a Mexican plant and is a poison of vegetable origin. It acts as a drastic purgative and also causes drastic congestion of the pelvic viscera, which may therefore have an effect on the uterus and cause a miscarriage. Such a drastic purgative, as jalap, can cause serious symptoms or even death – See Comrie, J.D., *Black’s Medical Dictionary*, p511

⁶⁹ Advocate, H.M. v. Lovie (1827) Syme’s Justiciary Reports, Appendix no. 2, p31.

On Tuesday the 14th of August 1827, Margaret developed symptoms of arsenic poisoning following a breakfast of porridge and died a few hours later.⁷⁰ Despite discussion of opening the body to carry out a post-mortem, Margaret was buried on Thursday the 16th August.⁷¹ Suspicion of murder could not be quelled however, and the Procurator Fiscal, after taking statements from Margaret's work colleagues and friends, made the decision that the body be subject to disinterment.

The body of Margaret was exhumed and a post-mortem carried out on Sunday 19th August by three surgeons from Fraserburgh in consequence of a warrant issued. Post-mortem examination revealed characteristics of arsenic poisoning.⁷² Various organs were sent to Dr. Blackie of Aberdeen and Dr. Christison of Edinburgh for chemical examination. Dr. Blackie carried out five different chemical tests from which he concluded that white oxide of arsenic was present in the body and had been the cause of death.⁷³ The testimony of Dr. Christison at trial corroborated this evidence, following the chemical tests that he had carried out.⁷⁴ Overriding opinion was that both the stomach and intestines of Margaret McKessor were saturated with arsenic.

During the trial all principal circumstances for establishing guilt were proved.⁷⁵ One of the most persuasive was undoubtedly the strong motive. In fact, despite Lovie's declaration in which he denied most of the principal evidence against him, all his statements at trial were proved false.⁷⁶ In addition, the integrity of the testimony of the Crown witnesses could not be dented by cross-examination, and the defence itself

⁷⁰ Large doses of arsenic commonly prove fatal in from 6 to 8 hours – See Taylor, A.S., *Medical Jurisprudence and Toxicology*, 12th edn, ed. A. Keith. Simpson, London, Churchill, 1965 p311.

⁷¹ Despite Margaret's relatives wishing her body to be opened Lovie persuaded them against this due to the scandal he said would arise when she was found with child – Advocate, H.M. v. Lovie (1827) Syme's Justiciary Reports, Appendix no. 2, p30.

⁷² Appendix 3, appearances -: 1, 5, 12, 13.

⁷³ Appendix 2, tests -: A5, A14, B3, B6, B10.

⁷⁴ Appendix 2, tests -: B2, B3, B9.

⁷⁵ Possession of poison by the accused, secret administration, motive.

⁷⁶ Lovie denied ever having had a relationship with Margaret, that he had known she was pregnant, that he had ever had any discussion with his ploughman about poisons and, added that he had bought arsenic not to kill rats but vermin upon black cattle – Advocate, H.M. v. Lovie (1827) Syme's Justiciary Reports, Appendix no. 2, p32.

called no witnesses. A more conclusive case could scarcely have been presented to a jury, but nevertheless a verdict of ‘Not Proven’ was given. Perhaps this was due to a jury’s inability to understand the medical evidence led at trial. It is more likely, however, that the returning of a ‘Not Proven’ verdict was due to the skilful oration of Lord Cockburn, who was Lovie’s counsel.

This acquittal is almost similar in circumstances to that of Mrs Smith.⁷⁷ Such acquittals seem hardly just, and, given both the ease of procuring arsenic and the general public interest in arsenic it is reasonable to suppose that this apparent unwillingness of juries to convict in such cases would have encouraged the perpetration of other such crimes, in the belief that there would be either non-discovery or a ‘Not Proven’ or ‘Not Guilty’ verdict obtained.

In 1827 a final trial took place, the eleventh case in this sequence. Here, Margaret Wishart in Arbroath was accused of poisoning her blind sister by placing arsenic in porridge the previous October. Wishart was found guilty and hanged on a gallows erected in front of the town hall.⁷⁸ Further details are absent from the records.

2.6 Mid-Century Profusion of Arsenic Cases

Between 1827 and 1837 very brief details for four arsenic poisoning cases exist. The twelfth case in this chapter concerns Alexander Wingate who murdered his mother and sister with arsenic in porridge. Both died and Wingate was found guilty and banished from Scotland.⁷⁹ In the thirteenth case George Frame was alleged to have poisoned his master, John Gilmour, and mistress Barbara Gilmour, in Renfrew by the common administration of arsenic in porridge. Both victims recovered and at his trial

⁷⁷ The histories of these cases are alike and in particular the existence of an unborn child. The only difference appears to be that in one case the accused was the mother of the seducer and in the other the seducer himself.

⁷⁸ Advocate H.M. v. Wishart Syme’s Justiciary Reports Appendix no.1 (1827), pp1-21. See also Christison, R., *EMJ*, vol 29, 1828, pp23-27, Cases and Observations in Medical Jurisprudence; Poisoning with Arsenic.

⁷⁹ See Record of the Lord Advocate AD14/31/228.

the jury reached a verdict of ‘Not Proven’.⁸⁰ The fourteenth case occurred in Fife and concerns Andrew Williamson, who attempted to poison his wife by putting arsenic in sugar. Williamson’s wife recovered and a ‘Not Proven’ verdict was reached at trial.⁸¹ In the fifteenth case Elizabeth Banks poisoned her husband Peter Banks in Edinburgh in 1835 by administering arsenic to him in salts and water. Peter died and Elizabeth was found guilty at trial and was hanged on the 3rd August 1835.⁸²

In contrast to the scant details of the above cases, the sixteenth case, of Elizabeth Jeffray (tried at Glasgow in April 1838), is noteworthy not only because there was an indictment libelling double murder, but for the fact that in this case a conviction was secured.⁸³ Mrs Jeffray lived in Carluke with her husband and the couple offered space for lodgers. In 1837, a young man from Skye, Hugh Munro, took up their offer of lodgings. At the same time a room in the house described as a “but and ben” was occupied by the widow Ann Carl and her nephew Walter Cullen.⁸⁴

At the start of October 1837 Mrs Carl became confined to bed. From the evidence of witnesses Mrs Carl partook of nothing at this point except tea prepared by Mrs Jeffray. She soon became so ill that she is reported to have said to a neighbour, Elizabeth Aitken that “she wished it was the Lord’s will to take her off this earth.” On the 4th of October 1837, Mrs Jeffray, with the help of Mrs Carl’s nephew, Walter, persuaded Mrs Carl to take a drink of whisky, meal and cream of tartar saying, “it was the only thing that would do her good.”⁸⁵ Soon after drinking the mixture Mrs Carl was seized with symptoms resembling arsenic poisoning.⁸⁶ She died early the next morning and was buried that same day.⁸⁷

⁸⁰ See Record of the Lord Advocate AD14/32/211.

⁸¹ See Record of the Lord Advocate Ad14/ 33/80.

⁸² See Record of the Lord Advocate AD14/35/437.

⁸³ Record of the Lord Advocate AD/14/38/275.

⁸⁴ But is the outer and ben the inner apartment of a two-roomed house – Hayward, A.L. & Sparkes, J.P., *Cassell’s English Dictionary*, p152.

⁸⁵ Record of the Lord Advocate AD/14/38/275

⁸⁶ The boy, Walter Cullen, who also had tasted the “mixture”, became unwell and vomited.

⁸⁷ This was at the insistence of Mrs Jeffray who said that “the body had a most disagreeable smell and she could not bear it.” – Record of the Lord Advocate AD14/38/275.

On the evening of Friday the 28th October 1837 Hugh Munro ate some porridge prepared by Mrs Jeffray and soon afterwards developed symptoms of arsenic poisoning. The symptoms continued throughout Saturday and Sunday when Mrs Jeffray gave Munro a powder which she said was rhubarb.⁸⁸ Following this Munro became worse and according to another lodger, Janet Meikle, “Munro was in a state of insensibility and foaming at the mouth.”⁸⁹ Despite Mrs Jeffray being asked to send for a doctor by Munro’s friend James McKay, she refused.⁹⁰ Munro died early on Monday morning and, at Mrs Jeffray’s insistence, was buried that same day.⁹¹

Suspicions having arisen, however, as to causes of death, both bodies were exhumed by direction of the Procurator Fiscal. Robert Logan, surgeon in New Lanark, carried out post-mortem examinations. Both examinations indicated arsenic poisoning.⁹² The viscera in both bodies were, therefore, removed by Dr. Logan and sent in sealed jars to Edinburgh for chemical analysis by Professors Traill and Christison. None of the results of chemical analysis appear to have been reported, but the opinions of all medical men were that death in both cases had been due to arsenic “present in the contents of the stomachs.”⁹³

With regards to possession of poison by Mrs Jeffray a strong inference of guilt was created at the trial due to the following circumstances. The witness, Jane Harkness, druggist in Carluke, stated that Mrs Jeffray had bought arsenic more than once from her prior to the deaths. “The first time she sent a little girl, Marion Tenant. Marion

⁸⁸ In small doses rhubarb is meant to be good for upset stomachs, about 40 grains mixed with soda – Comrie, J.D., *Black's Medical Dictionary*, 8th edn, A & C Black Ltd, London, 1926, p765

⁸⁹ Record of the Lord Advocate AD14/38/275.

⁹⁰ Mrs Jeffray’s reasons for not sending for a doctor were twofold. Firstly she stated that Munro would object to the expense. Secondly that she would have no doctor in her home but Dr. Rankine, who was not at home – Record of the Lord Advocate AD/14/38/275.

⁹¹ Once again Mrs Jeffray gave the same reason that she had used for Mrs Carl, that the body had a disagreeable smell and she could not bear it – Record of the Lord Advocate AD14/38/275. Noteworthy is that both in the case of Mrs Carl and Hugh Munro Mrs Jeffray had refused to look at the bodies after death. Such behaviour constituted, in Scotland at that time, a gross breach of courtesy to the dead.

⁹² In the case of Mrs Carl, see Appendix 3, appearances -: 1, 4, 5, 11. In the case of Hugh Munro, see Appendix 3, appearances -: 1, 2, 3, 5, 13.

⁹³ Record of the Lord Advocate AD/14/38/275.

bought threepence worth of arsenic and said it was for Mrs Jeffray.” Further, on the Friday before Munro’s death, Mrs Jeffray herself purchased a half-ounce of arsenic saying she required it for the purpose of poisoning rats.⁹⁴ Mrs Jeffray then returned for a further supply of arsenic on the day before Munro died, saying she had lost what she had previously bought. Indeed, additional suspicion of guilt was inferred at trial by Mrs Jeffray’s attempts to account for her possession of arsenic by false statements.⁹⁵

The prosecution during the trial made no attempt to attribute to Mrs Jeffray any motive for poisoning Mrs Carl.⁹⁶ As for the death of Munro, the motive appears to have been pecuniary gain. Hugh Munro had entrusted £5 to Mrs Jeffray whilst he worked away during the sheep shearing season. Indeed, on Munro’s return, Mrs Jeffray stated her inability to repay his money and following his death that she had restored it to him.⁹⁷ From evidence, however, it would appear that the repayment story was untrue. The defence in this case relied heavily on the rat excuse, which had already been proved false. Following a trial of eighteen hours the jury, by a majority, found the panel guilty and she was executed on May 21st 1838 for double murder.⁹⁸

Of interest is the fact that before living in Carluke, Elizabeth Jeffray had run a lodging house in Bathgate. There, she was considered by neighbours to be a particularly malicious character who, bearing a grudge against a neighbour, was reputed to have poisoned her pig. Moreover, one of her Bathgate lodgers was reputed to have died

⁹⁴ Mrs Jeffray said to the druggist that “she had killed one rat with the first quantity purchased and she wanted to try it again.” – Record of the Lord Advocate AD14/38/275. On this occasion Mrs Jeffray also bought 3d worth of laudanum which she said was for a sick lodger who had cholera.

⁹⁵ No evidence was given during the trial to show that Mrs Jeffray had ever used arsenic to destroy rats, and no sufficient proof was given of there being rats in the house.

⁹⁶ Unless it was perhaps to try out the poison which was later to be administered to her other victim.

⁹⁷ It is noteworthy; however, that Mrs Jeffray’s daughter was married whilst Munro was away working during the sheep shearing season, with no expense spared. The equivalent value of £5 in today’s money is roughly £365, which would have gone an extremely long way in 1837. See Record of the Lord Advocate AD14/38/275.

⁹⁸ For some strange reason, however, the jury unanimously recommended Mrs Jeffray to mercy. Advocate, H.M. v. Jeffray (1838) 7 Swinton 113, p133.

suddenly and in suspicious circumstances.⁹⁹ Perhaps these episodes could be regarded as preliminary affairs for which she was brought to trial and indeed it seems possible that such a woman may have previously used arsenic.

My seventeenth case occurred in 1839 although little detail exists. In 1839 in Banff, James Mellis was charged with attempting to murder his masters Charles Hay and George Hay by putting arsenic in broth. Mellis was employed as an apprentice tailor and the brothers, having no servant, habitually prepared their own meals. Having partaken of the broth both brothers became seriously ill, but recovered fully. Arsenic was found in the broth, but at trial the jury reached a ‘Not Proven’ verdict against Mellis.¹⁰⁰

The 1840s appear to have been the peak period in the use of arsenic both in Scotland and England with weight being given to this by both my figures and the findings of other writers.¹⁰¹ My findings show, however, that the 1830s did not lack very far behind¹⁰² and it is possible to say that the mid nineteenth century was a time period when arsenic poisoning in Scotland was at its peak.

The eighteenth case and the first case in the 1840s concerned a certain Mary MacFarlane or Taylor who faced trial in Glasgow in 1843 for the murder of both her husband and a neighbour by administering arsenic. Both died, but there was an acquittal during trial due to legal complexities that arose.¹⁰³

The nineteenth case in the sequence concerns the trial of Christina Gilmour in 1844 for the murder of her husband with arsenic. The surprising ‘Not Proven’ verdict

⁹⁹ Record of the Lord Advocate AD14/39/28.

¹⁰⁰ See Record of the Lord Advocate AD14/39/28.

¹⁰¹ 21% of all my arsenic cases occurred in the 1840s. See also Whorton, J.C., *The Arsenic Century: How Victorian Britain was Poisoned at Home, Work and Play*, Oxford University Press, 2010, p25; Burney, I., *Poison Detection and the Victorian Imagination*, Manchester University Press, 2006, p20; Bartrip, P.W.J., *Medical History*, vol 36, 1992, pp53-69, A Pennurth of Arsenic for Rat poison: The Arsenic Act of 1851 and the Prevention of Secret Posioning.

¹⁰² 18% of all my arsenic cases occurred in the 1830s.

¹⁰³ See Cockburn, H, *Circuit Journeys*, David Douglas, Edinburgh, 1888, p190.

attests to the fact that little legal advances had been made since the early years of the century. In 1842, Christina, under parental pressure, had married John Gilmour, a farmer, and moved with him to his farm at Inchinnan near Renfrew. On arrival at the farm Christina had announced to family and friends that she would never live with her husband as his wife.¹⁰⁴

On December 26th 1843, after a year of marriage, Christina ordered Mary Paterson, a servant, to purchase “twopence worth of arsenic to kill rats.”¹⁰⁵ Accordingly Mary purchased the arsenic and gave it to her mistress. On the 29th December 1843 John Gilmour took ill with symptoms characteristic of arsenic poisoning. His wife, on January the 6th 1844, went to Renfrew to allegedly purchase “something that would do her husband good.”¹⁰⁶ However, on her return she dropped her purchases, which were seen by a farm hand and a maid; both noted that included was a paper packet marked “poison.” The following day a young lady giving the name ‘Miss Robertson’ of Paisley obtained from a Renfrew chemist twopence worth of arsenic, upon the usual pretext. John Gilmour grew gradually worse and was attended by a Dr. McKechnie of Paisley who was suspicious that the man’s illness might not be attributable to innocent causes.¹⁰⁷ On the 11th January 1844 John Gilmour died, expressing a wish before he did so, “to be opened” and saying “oh, that woman – if you have given me anything tell me before I die.”¹⁰⁸

Following the funeral, Christina returned to her parents, resuming her friendship with her first love. However, three months later in consequence of many rumours and a warrant issued by the local sheriff, the body of John Gilmour was exhumed and examined by Drs Wylie and McKinlay from Paisley. Post-mortem appearances were

¹⁰⁴ At that time Christina had fixed her heart upon another – Advocate, H.M. v. Gilmour (1844) Brown 23, p25.

¹⁰⁵ Advocate, H.M. v. Gilmour (1844) Brown 23, p27.

¹⁰⁶ Ibid, p28.

¹⁰⁷ Being suspicious Dr. McKechnie asked Christina to preserve for examination all vomited matter. Calling next day he asked for this, but was told by Mrs Gilmour that there was so little that it had not been worth keeping.

¹⁰⁸ Advocate, H.M. v. Gilmour (1844) Brown 23, p28.

characteristic of arsenic poisoning and the viscera were removed for further testing.¹⁰⁹ From eight different tests carried out, both doctors concurred that not only was arsenic present, but that it had been the cause of death.¹¹⁰ Dr. Christison in Edinburgh made further experiments upon other portions of the viscera and he corroborated the opinion of the other two doctors.¹¹¹ This was the first case in which Reinsch's test was used for medico-legal purposes in Scotland.¹¹²

When the case came to trial, the facts of the medical evidence indicated the possibility of simple inference that John Gilmour had died from repeated doses of arsenic. With regards possession of poison the evidence was clear that Christina had been in possession of three packets of arsenic and that she had made repeated purchases of arsenic in a secretive manner, in the last instance by means of false statements and pretences. Indeed, during the trial, three witnesses identified Christina as being the unknown purchaser 'Miss Robertson'.¹¹³ In addition, Christina had ample opportunity to administer arsenic to her husband.¹¹⁴

The above being proved, presence or absence of motive at trial had to be considered. Clearly the motive for carrying out this crime was Christina's dissatisfaction with her marriage and noteworthy is her immediate return to her old love following the death of her husband.¹¹⁵ During the trial the defence maintained that John Gilmour had poisoned himself, but this assertion received little support from the collated evidence. Indeed, the defence called no witnesses. Following a damning speech by the Lord Advocate, and a sentimental address from the defence, the jury reached a unanimous

¹⁰⁹ Appendix 3, appearances :- 1, 2, 3, 4, 5, 6, 9, 13.

¹¹⁰ Appendix 2, tests :- A5, B2, B3, B9, B10, B11.

¹¹¹ Appendix 2, tests :- B10, B11, B12, B16.

¹¹² Hugo Reinsch's test was published in 1841; see Appendix 2, B. 12. See also Cambell, W.A., *Chemistry in Britain*, vol 1, 1965, pp99 –201, Some Landmarks in the History of Arsenic Testing.

¹¹³ *Ibid.*

¹¹⁴ In the house where the couple had lived no servant attended indoors and Christina had sole charge of preparing food etc. She, therefore, had every opportunity to mix arsenic in her husband's food. Also no satisfactory account was given for disposal of the arsenic she had bought.

¹¹⁵ In the statement contained in the prisoner's own declaration Christina maintained she had bought poison for the purpose of dissolving her marriage by committing suicide – Advocate, H.M. v.Gilmour (1844) Brown 23, p35.

verdict of ‘Not Proven’. This occurred despite all the principal circumstances for establishing guilt being present.¹¹⁶

It is possible that there existed unwillingness for juries during the nineteenth century to convict a woman on a charge of poisoning on evidence which was merely circumstantial. Indeed, in the trial of Janet Campbell or McLellan, the twenty-second case, in Edinburgh two years later for the murder of her husband with arsenic an actual ‘Not Guilty’ verdict was reached.¹¹⁷ This was despite it being proved that she had on two occasions purchased arsenic and had given birth to twins as a result of an affair with a lodger. In addition, arsenic was found “to a considerable extent” in the body of the deceased by Dr. Thomson of Perth and Professor Christison.¹¹⁸ Consideration should, in light of this and the Gilmour case, be given to the question of why was Mrs Jeffray was found guilty. Upon similar medical evidence, directly opposite verdicts were reached which calls the reliability of juries into question. It is worth considering also that Jeffray was by birth English, and so a foreigner, and, in contrast to Mary Smith, Christina Gilmour and Janet Campbell, was arraigned with narrative testimony of possessing a malicious disposition, was unpopular and unattractive. The case-by-case evidence cannot rule out such subjective components of juries’ decisions.

Between 1844 and 1850 a further four cases are present in the records. The twentieth case is that of Janet Hope in Lockerbie who was charged with the murder of her son in 1845, but acquitted.¹¹⁹ In the twenty-first case James McKerlie was charged with attempting to murder three others with arsenic in 1845. All parties recovered although McKerlie was found guilty and transported for life.¹²⁰ The twenty-third case occurred in Aberdeen in 1849 and involved a forty- four year old farm servant, James Burnett, who was found guilty and executed for the murder of his wife of twenty-six years by placing arsenic in her porridge.¹²¹ The twenty- fourth case saw Margaret Hamilton

¹¹⁶ Possession of poison, secret administration, motive.

¹¹⁷ Advocate, H.M. v. Campbell (or McLellan) (1846) Arkley 137, p137.

¹¹⁸ Ibid, p169.

¹¹⁹ Advocate H.M. v. Hope (or Tedcastle) Broun II (1844-1845), pp465-468.

¹²⁰ Advocate H.M. v. McKerlie Broun II (1844-1845), pp429-442.

¹²¹ See Record of the Lord Advocate AD14/49/256.

hanged in Glasgow on the 31st January 1850 for the murder of her sister with arsenic in Strathaven in 1849.¹²²

By the 1850s there began to be a slow, but steady decline in the number of reported arsenic cases in Scotland. Arsenic now represented only 15% of all poisoning cases as compared to the figure of 21% during the 1940s. By the end of the nineteenth century this figure had fallen as low as 6% as medical jurisprudence had became more of a disciplined subject and chemical testing was less hit and miss and with an increased degree of certainty.

The first case in the 1850s, the twenty-fifth case, occurred in July of 1850, when William Bennison was tried in Edinburgh for the murder of his wife Jean to whom he had been married for eleven years. The couple lived at the bottom of Leith Walk in a small cul de sac named Stead's Place. On March 14th 1850 Bennison visited a druggist, William McDonald, and purchased an ounce of arsenic stating he wished to kill rats. Notwithstanding that Bennison was supposed to be a devout Methodist it would appear he administered arsenic to his wife on Friday 12th April 1850 in her evening meal of porridge. Jean soon developed symptoms of arsenic poisoning and on Sunday 14th April, Bennison fetched her sister Helen, telling her to "come quickly if you wish to see your sister in life." Helen on reaching the Bennisons' house in Stead's Place concluded that her sister was indeed dying and so remained with her. Just before midnight Jean requested a doctor, but Bennison answered that "there was no use incurring expense as she was dying." Indeed, during his wife's last hours Bennison prepared the burial clothes and wrote funeral letters so that final arrangements would be well in hand. Jean died early on Monday 15th of April after having called out that she was "waiting for the coming of Jesus." Burial took place on April 18th and, on the same day Bennison revisited the druggist William McDonald, to plead with him not to mention to anyone that he had purchased arsenic. His reason for this, he said, was that such talk would harm him with the authorities.¹²³

¹²² Young, A.F., *The Encyclopaedia of Scottish Executions*, Eric Dobby Publishing Ltd, 1998, p117.

¹²³ Record of the Lord Advocate AD14/50/488

According to neighbours, Bennison had been seen often in the company of a young woman, Margaret Robertson, before his wife's death. He had also often appeared restless and agitated before the death of Jean. Jean's sister accordingly became suspicious and wrote to the Procurator Fiscal.¹²⁴ Jean's body was exhumed on April 24th, by direction of the Procurator Fiscal, and examined by Doctors Maclagan and Anderson of Edinburgh.¹²⁵

During the trial of William Bennison, Dr. Maclagan gave evidence to show that post-mortem appearances were characteristic of arsenic poisoning.¹²⁶ Also that following Reinsch's test both the stomach contents and liver gave positive results for the presence of arsenic.¹²⁷ Arsenic was further detected by Reinsch's process in matter vomited by Jean, and further, in the pot in which her last meal of porridge had been made. Death in this case was clearly attributed to arsenic poisoning, with the medical evidence being corroborated by Doctors Anderson and Spittal of Edinburgh.

During his trial, proof of possession of poison by Bennison, was deemed unequivocal. In addition, every opportunity had existed for secret administration. With regards to motive, it would appear that this was twofold, Bennison's growing fondness for Margaret Robertson was attested to and the financial gain which resulted from his wife's death was also noted. Upon Jean's death, Bennison received £11 from various benefit societies of which he had been a member. In fact, Bennison had previously boasted to one witness of the financial benefits he would receive on the death of his wife – "Members would have about £3 from each of the two yearly societies and more from the foundry on the death of a wife."¹²⁸ The jury unanimously found Bennison guilty during trial and sentence was passed that he be executed.¹²⁹

¹²⁴ Rumours had begun to spread that Mrs Bennison had been poisoned following the deaths of two neighbour's dogs, which had eaten cooked potatoes put out by Bennison on the night of his wife's death – Record of the Lord Advocate AD14/50/488.

¹²⁵ Dr. Maclagan eventually became Sir Douglas Maclagan.

¹²⁶ Appendix 3, appearances :- 1, 2, 4, 5, 12.

¹²⁷ Appendix 2, test :- B12.

¹²⁸ Record of the Lord Advocate AD/14/50/488. Bennison was employed at the local Shotts Iron Foundry in Leith Walk, Edinburgh.

¹²⁹ Record of the Lord Advocate AD/14/50/488.

The passing of the verdict was made more likely by the emergence during trial of the fact that Bennison had bigamously married Jean.¹³⁰ In 1839, shortly after the purported marriage to Jean, Bennison had brought his first wife to Airdrie where she fell sick and died. During the trial for the murder of Jean a substantial amount of evidence was uncovered which rendered it probable that Bennison had also caused his first wife's death by poisoning her with arsenic. Thus, while never tried for this offence, it represents another possible case of murder by arsenic to add to the mass of unrecorded, even completely undetected cases, which the current sequence suggests must surely exist in Scottish history.

The twenty-sixth and twenty- seventh cases, concern a joint charge in 1852, involving a woman, Sarah Fraser, who, along with her son, James, poisoned her husband for pecuniary gain by putting arsenic in his porridge. Fraser senior died, but at trial, due to unreliability of evidence - a sealed paper wrapper containing powder was discovered to have been tampered with after the arrest - the pair walked free.¹³¹

2.7 The Arsenic Act and Reluctance to Convict

Justice in the Victorian period, as the panoply of cases above demonstrate, could certainly be described as being frustrated by incompetence or bias. Nevertheless, while such bias persisted throughout the century, the passing of the Arsenic Act in 1851 marked a shift in juridical and public attitudes to the legal and social relationship to arsenic and to sentencing for arsenic poisoning. Where previously arsenic purchase had been unregulated, the mid-century Act made clear that this was now seen as dangerous and socially unacceptable. Previously society had simply put up with this laissez-faire status quo and was content to punish those proved to abuse their freedom to purchase arsenic, by sending offenders to the gallows. However, attitudes were changing in the mid-nineteenth century. Where once crowds had flocked to see public hangings, more humane ideals began to take root and the beginning of a reluctance to

¹³⁰ Bennison had married a Mary Mullen in 1838 in Ireland and as it was a valid marriage he was also charged with bigamy.

¹³¹ Advocate H.M. v Fraser Irvine (1852) 1 Irvine 1

sentence any criminal to death could be noted.¹³² Hangings ceased to be public in Glasgow in 1865.¹³³ Indeed, of the eight trials for murder or attempted murder by arsenic poisoning after the passing of the 1851 Arsenic Act, only one resulted in a guilty verdict.¹³⁴

The limitations upon the serving of justice which have been consistently shown in the small cases which concern this study are also apparent in the larger scale and more famous trials of the period, such as that of Madeleine Smith in July 1857 for the murder of Emile L'Angelier with arsenic.¹³⁵

Such is the notoriety of this case that it cannot be passed over, even in this present enquiry which seeks to elucidate overall trends from the mass of lesser known trial records. Madeleine Smith was the daughter of a wealthy Glasgow architect who had fallen in love with a shipping clerk, twenty six year old Emile L'Angelier whom she had met by chance one day whilst out walking. The couple became lovers and even addressed one another as “husband” and “wife” in correspondence. An elopement was anticipated, but Madeleine, knowing that L'Angelier's salary of only ten shillings a week would not keep her in the manner to which she was accustomed, encouraged the attention of a wealthy merchant who lived in the flat above hers, and, on the 28th January 1857, accepted his hand. Madeleine then began to demand the return of letters that she had sent to L'Angelier and upon his refusal and his threats to pass the letters to her father; she poisoned him by lacing a cup of cocoa with arsenic.

During the trial, her lawyers used the Styrian defence, arguing that L'Angelier could have been a secret arsenic eater, and that Madeleine had bought arsenic to use as a

¹³² During the early reign of Queen Victoria (1837-1850) huge crowds attended public hangings. Indeed, schools were dismissed early to allow male pupils to witness hangings and rooms giving a good view of the scaffold rented out.

¹³³ The last public hanging in Glasgow was that of Dr Pritchard on 28th July 1865.

¹³⁴ See trial of Ellen McLeod or Beaton in 1896; case number 34, which resulted in a sentence of 15 months- Record of the Lord Advocate AD/14/96/95.

¹³⁵ See Tennyson, J.F., *Trial of Madeleine Smith*, Notable British Trials, W. Hodge & Co. Ltd, Edinburgh & London, 1927.

cosmetic.¹³⁶ A ‘Not Proven’ verdict was reached by the jury and Madeleine walked free. Similar biases to those noted in the case of Mrs Jeffray have been noted in the case of Madeleine Smith. While the former’s malice and ugliness seem to have counted against her, the latter’s good looks and her position of respectability would appear to have contributed to her acquittal. Indeed, John Inglis, counsel for the defence, played heavily on these subjective themes in attempting to portray Smith’s character in a positive light. Virulent anti-French sentiment during the 1850s has also been taken into account. The verdict of the jury may, therefore, have been more a posthumous condemnation of L’Angelier than one of genuine doubt.¹³⁷

The twenty-ninth case in this sequence occurred in 1859 and concerns the murder by David Ross of his brother Walter Ross by putting arsenic in gruel for financial gain. A ‘Not Proven’ verdict was returned at trial however by a majority of one.¹³⁸ Case thirty involves a young servant, Agnes Kirkwood, who was charged in June 1861 with attempting to murder her master and his son by putting arsenic in their porridge.¹³⁹ Once again the setting was a farm, although in this case no circumstances existed to indicate purchase of poison by the accused. Rat poison, however, in the form of Kearney’s Powders, was kept in the kitchen and accessible to all.¹⁴⁰ On the 17th June 1861 both father and son took ill with symptoms of arsenic poisoning. No doctor

¹³⁶ See Emsley, J., *The Elements of Murder*, Oxford University Press, 2005, pp158-159 & Roughead, W., *Classic Crimes*, New York Review Books, New York, 2000, pp129-169.

¹³⁷ See Altick, R.D., *Victorian Studies in Scarlet*, L M Dent & Sons Ltd, London, 1972, p179. It is difficult to overestimate the extent to which the British, after the defeat of Napoleon in 1815, continued to feel paranoia about France. Not only did all the British military, and many of their politicians, continue to believe that the greatest political threat came from France (up to and even during the Crimean War which ended in 1856 and when French and British were supposedly allies); not only did prime ministers Palmerston and Wellington fear the prospect of French invasion long after the possibility of such an event had been extinguished; but France was also seen during the nineteenth century as the very object of what could happen if a society imploded - See also Wilson, A.N., *The Victorians*, Hutchinson, London, 2002, pp16-17.

¹³⁸ Advocate H.M. v. Ross Irvine vol 111 (1858-1860), pp434-439.

¹³⁹ Record of the Lord Advocate AD/14/61/79.

¹⁴⁰ The Arsenic Act of 1851 did not prohibit the purchase of such products containing arsenic.

attended, but various items were sent to Dr Maclagan of Edinburgh for testing.¹⁴¹ Using Reinsch's method Dr. Maclagan detected arsenic in both vomited matter and porridge.¹⁴² Despite unequivocal proof of the presence of arsenic in the matter tested, little further circumstantial evidence existed at trial, apart from the motive of revenge.¹⁴³ The jury, therefore, found Miss Kirkwood 'Not Guilty'.¹⁴⁴

In the thirty-first case, that of Alexander Paterson in 1869, the charge was one of culpable homicide rather than murder or attempted murder. Alexander Paterson had originally been a gardener and developed an interest in meddling with and recommending various medicines. He prescribed and applied on various occasions an ointment containing arsenic to the inflamed breast of a Mrs L and as a consequence arsenic was absorbed into her system and she died. At trial Paterson was found guilty, but a lenient sentence recommended and he only served four months.¹⁴⁵

The thirty-second case again is a prime exemplar of the lenient attitude towards inflicting death through use of arsenic, which would appear to have existed in the late nineteenth century. The trial concerns John Webster, landlord of the Newton Hotel, Kirremuir. Webster was tried at Edinburgh in February 1891, for the murder of his wife with arsenic.¹⁴⁶ Early August 1890 Mrs Webster became ill with symptoms of arsenic poisoning. She died within three days, and despite an initial diagnosis of gastro-enteritis by the local doctor, suspicions led to a warrant being issued to exhume the body, albeit four months after the death.

¹⁴¹ Suspicion had arisen after Mr Kincaid fed some of the porridge, which he and his son had eaten, to a hen. The hen subsequently died and was found to contain a substantial amount of arsenic following testing by Dr. Maclagan of Edinburgh.

¹⁴² Appendix 2, test -: B12.

¹⁴³ Agnes could have been seeking revenge for loss of permanent employment on the farm. She had been most angry about this, perhaps providing a motive for her to seek avengement.

¹⁴⁴ Record of the Lord Advocate AD/14/61/79.

¹⁴⁵ See Graham, T., *Half Yearly Abstract of the Medical Sciences*, vol XLIX, 1869, pp118-121.

¹⁴⁶ Roughead, W., *Glengary's Way and Other Stories*, W. Green & Son Ltd, Edinburgh, 1912, pp113-114.

A post-mortem examination carried out by Dr. Harvey Littlejohn revealed characteristics of arsenic poisoning.¹⁴⁷ Noteworthy also was the unusually well preserved state of the body and that there were no signs of natural death. Indeed, during trial, evidence was given by Dr. Littlejohn to the effect that both Marsh's test and Reinsch's test in all organs examined had detected arsenic.¹⁴⁸ This was corroborated by testimony of Dr. Falconer King and Professor Crum Brown of Edinburgh. Death had been due to arsenic, administered in repeated doses. Despite no circumstances existing to prove purchase or possession of poison by the accused, motive was particularly strong. It was proved that not only had the accused and his wife been on very bad terms, but that Mr Webster had insured his wife's life for £1,000 shortly before her death. The defence argued at trial that the arsenic in Mrs Webster's body had been due to her taking Fowler's Solution.¹⁴⁹ It was, however, not proved that she had ever taken this nor was any found in the house.¹⁵⁰ Following a three-day trial, the jury gave a verdict of 'Not Guilty' and Mr Webster walked free.

The thirty-third and final trial within my time period is that of Ellen Mcleod or Beaton who tried to poison her master Thomas Hay and three other farm servants by putting arsenic in broth. Once again Kearney's Powders for killing rats was used. All victims survived however and Ellen was sentenced to fifteen months imprisonment.¹⁵¹

Although arsenic poisoning ceased to be common after the nineteenth century the arrival of the twentieth century did not see an end to poisoning with arsenic. During a silver wedding anniversary dinner party held on the 3rd February 1911 by a Mr and Mrs Hutchison of Dalkeith, their son John Hutchison put arsenic in the coffee served to guests with the result that two of the guests died. There was no trial in this case as John Hutchison, on being charged with the lesser crime of culpable homicide and questioned by a police sergeant, ran to his bedroom and swallowed the contents of a

¹⁴⁷ Appendix 3, appearances :- 1, 2, 5, 8, 10, 12, 13.

¹⁴⁸ Appendix 2, tests :- B10, B11, B12.

¹⁴⁹ A patent medicine which was used as a tonic and contained arsenic.

¹⁵⁰ Note that there is only one recorded case in which Fowler's Solution has destroyed life. This was in 1848 – Taylor, A.S., *Medical Jurisprudence and Toxicology*, 12th edn, ed. Keith. Simpson, Churchill, London, 1965. p308.

¹⁵¹ See Record of the Lord Advocate AD14/96/95.

phial of prussic acid, resulting in his death within a few seconds.¹⁵² Further to this, in 1924, William Laurie King was tried at the High Court in Edinburgh for the murder of his mother and attempted murder of his father with arsenic.¹⁵³ Not since the trial of Madeleine Smith had arsenic trial involved such a respectable family.¹⁵⁴ Under parental pressure William had begun an apprenticeship with a firm of chartered accountants in 1920.¹⁵⁵ However, desiring to follow a scientific career, William felt great resentment towards his parents and poisoned them by putting arsenic in the family supper of bread and cheese. This trial is of interest due to the fact that it appears to be the first poisoning trial in Scotland in which circumstances of proving possession of poison were met with the admittance of real evidence.¹⁵⁶ Indeed, from the outhouse and attic that William used to pursue his photography interest were removed eighty-three bottles of chemicals including arsenic.¹⁵⁷ During the trial, defence counsel for William argued that his mother had died by poisoning herself from amongst the twenty-one medicines removed from her bedroom.¹⁵⁸ The jury were therefore reluctant to convict and William was found ‘Not Guilty’.

2.8 Trends and Conclusions

Significant and constant in most of these cases is the frequently used “rat-excuse”. Indeed, the “rat-excuse” was used in twenty-one of these trials (66%). Also notable

¹⁵² Roughead, W., *Glengarry’s Way and Other Stories*, W. Green & Sons Ltd, Edinburgh, 1912, pp115-7.

¹⁵³ Record of the Lord Advocate AD/15/24/22.

¹⁵⁴ Mr King senior was an accountant and lived with his family in a very affluent area of Edinburgh- 2 Wester Coates Terrace.

¹⁵⁵ Messrs. Brewis, Rainie and Boyd – 6 Daingway Street. William later transferred to work at his father’s firm.

¹⁵⁶ Real evidence may be considered as “a thing which may be a human being, any feature of the thing which may be significant, and the inferences to be drawn from the existence of the thing or from its significant features.” – Walker, A.G. & Walker, N.M.L., *The Law of Evidence in Scotland*, W.Green, Edinburgh, 1964, para416. Note real evidence must generally be accompanied by oral evidence from a witness with first hand knowledge of the item.

¹⁵⁷ Of interest is the fact that the outhouse was not in fact large enough to store all of William’s chemicals and so some were kept in the house attic – Record of the Lord Advocate AD/15/24/22.

¹⁵⁸ These included Fowler’s Solution, which contained arsenic and an ointment of iron and arsenic to deal with toothache.

throughout the period is the ease with which arsenic could be obtained by any person of any class or occupation. Despite the Arsenic Act of 1851 and the important Pharmacy and Poisons Act of 1868, many arsenic-containing treatments, solutions and remedies were still widely available.¹⁵⁹ Of note also is that many of the cases occurred in rural areas – essentially arsenic was a way of settling grievances or profiting from the death of another, with little risk of punishment given the patchy nature of the medico-legal establishment in the backwaters of the nation.¹⁶⁰

Remarkable is the fact that out of these thirty-two poisoning trials, eighteen of the accused walked free (56%).¹⁶¹ In many cases however, the circumstances in these trials pointed towards guilty verdicts. Why, therefore were such dubious verdicts reached? It is possible that part of the answer to this falls within the field of forensic toxicology in relation to medical jurisprudence. During the nineteenth century medical jurisprudence became an increasing object of interest. The chief difficulty, however, in the presentation of toxicological evidence during a poisoning trial was its perplexing, inaccurate and unsophisticated systems of classification. The medical jurist had to determine first, whether the crime of poisoning had been committed and secondly, what particular poison had been employed.¹⁶² Indeed, in a trial where so much of the evidence was circumstantial, it was necessary to have ample proof of the cause of death, and with no distinctive diagnostic symptoms for arsenic poisoning diagnosis was often most difficult. Evidence against an accused, therefore, depended largely upon expert medical testimony, with the role of the jury being as an arbiter of fact.

¹⁵⁹ The Pharmacy and Poisons Act of 1868 attempted to confine the sale of a range of poisons to qualified pharmacists.

¹⁶⁰ This is not surprising given that until 1851 only 22% of the Scottish population lived in the four big cities of Glasgow, Edinburgh, Dundee and Aberdeen. Indeed, even by 1911 this figure had only risen to 30%.

¹⁶¹ 11 ‘Not Proven’ verdicts, 3 ‘Not Guilty’ verdicts and 4 Acquittals. In 4 of the 18 cases (22%) the accused owed their immunity from punishment to some loophole in the law. These technical acquittals included (1) & (2) Sarah Fraser and James Fraser – Advocate, H.M. v. Fraser (1852) 1 Irvine 1; (3) Mary Taylor or MacFarlane – Advocate, H.M. v. Taylor (or MacFarlane) (1843) 1 Broun 550; (4) Janet Hope or Walker – Advocate, H.M. v. Walker (or Hope) (1845) 2 Broun 429.

¹⁶² They had to consider the symptoms, the morbid appearances and also the natural diseases, which imitate these symptoms and appearances, and the effects of the suspected material on animals.

Such trials with their adversarial methods and requirement of admissible legal evidence, and the need to exclude the reasonable possibility of accident or death from illness, must frequently have confused the jury to the point that they were unable to unequivocally recommend ‘guilty’ verdicts. Some doctors proved themselves so well versed in medical matters relating to arsenic poisoning, that they often overlooked the jury’s considerable lesser knowledge. In addition, the requisite science was confined to only a few, and often during trials evidence was given by other doctors who, despite displaying detailed textbook knowledge of arsenic poisoning, had little or no practical experience within this field. Such medical men might act in a hesitating manner and show unsatisfactory knowledge in the witness box, possibly bewildering juries even further.¹⁶³ In addition, many of the chemical tests employed to detect arsenic required considerable analytical skill, and samples could very easily become contaminated.¹⁶⁴ The complexity of the Marsh test made it difficult for amateurs to use and many medical practitioners had little or no experience of what to look for during an autopsy. Further, there was a constant bickering amongst prominent medical men on issues relating to the validity of medical evidence given in poisoning trials.¹⁶⁵

It is, therefore, of little surprise that medical evidence was generally distrusted in courts of law during the nineteenth century.¹⁶⁶ Indeed, during the mid nineteenth

¹⁶³ In particular see Advocate, H.M. v. Elder (or Smith) (1827) Syme 71, pp71-131 – The Lord Advocate: “Dr. Mackintosh you have heard the evidence of arsenic being found in the stomach of the woman. Are you satisfied that arsenic was discovered there?” – Dr. Mackintosh: “My Lord, I am no judge of chemical evidence.” – The Lord Advocate: “Suppose arsenic was detected, what in that case do you think was the cause of these symptoms and of these signs in the dead body?” – Dr. Mackintosh: Natural disease might cause them all.”

¹⁶⁴ This was in particular when body tissues and fluids were involved.

¹⁶⁵ See in particular: Christison, R., *EMJ*, vol 27, 1827, Account of the Medical Evidence in the Case of Mrs Smith, Tried at Edinburgh in February Last for Murder by Poison, pp441-472; Gamgee, A., *EMJ*, vol 10, 1864-1865, On an Alleged Fallacy in Marsh’s Process for the Detection of Arsenic, pp409-415.

¹⁶⁶ In the words of the Lord Advocate in the trial of Mary Elder or Smith: “... but there were uncertainties - were blunders- and it was the pride of one age to rear up theories to be trampled down and triumphed over by the next.” – Advocate, H.M. v. Elder (or Smith) Syme 71, p128.

century there was great concern among British scientists about the role of the expert witness, particularly with regard to the effect of the legal constraints on the validity of scientific evidence. It was argued that to put a scientist in the position of an advocate was “far removed from the idea of a man of science” and indeed scientific evidence only provided a certain degree of probability and could never achieve the incontrovertible demonstrations of proof required by the courts.¹⁶⁷ Further to this was the inability of the judiciary to understand the fundamentals of scientific reasoning and how it differed from legal argument. It is thus not surprising that so many juries acquitted persons who may appear today to be clearly guilty of the crime of murder or attempted murder by poison, since while having no real doubt of guilt; juries did not believe the case against a panel to be legally proven.¹⁶⁸

The outcome of such trials as have been investigated above may have also been due sometimes to the ignorance of the medical profession with respect to arsenic poisoning. It is very possible that diagnosis of arsenic poisoning was too difficult for medical men in the nineteenth century, especially those lesser-experienced practitioners working in rural areas, since the leading characteristics of arsenic poisoning were very similar to cholera, gastro-enteritis, spontaneous erosions of the stomach, strangled hernia, peritonitis, and melanaemia.

Certainly, as has been noted, legislation did little to counteract the unrestricted selling of arsenic.¹⁶⁹ Juridical tendencies and limitations restricted the possibility of conviction even if poisoning was suspected or detected and, as several of the above cases hint, many of the defendants may have often committed similar poisoning offences. The plethora of arsenic poisoning cases thus, is highly likely to have been

¹⁶⁷ Hamlin, C., *Social Stud Science*, vol 13, 1986, Scientific Methods and Expert Witnessing, Victorian Perspectives on a Modern Problem, pp485-513.

¹⁶⁸ Such acquittals were particularly prevalent during the middle of the period (1837-1901); where out of 17 poisoning trials, 10 (59%) resulted in a ‘Not Proven’ verdict, ‘Not Guilty’ verdict or technical acquittal. This surely reflected not only confusion among juries but highlighted a definite change in attitude towards matters of life and death. Between 1800 and 1837 there had been 8 acquittals in 17 trials (47%) – 6 ‘Not Proven’ verdicts and 2 ‘Not Guilty’ verdicts. Surprisingly in the whole period that I have covered (1800-1940) there is little difference in the number of acquittals for men and women :- 34 trials resulted in 10 acquittals for women (29%) and 8 for men (24%).

¹⁶⁹ See Paris J.A., & Fonblanche, J.S.M., *EMJ*, vol 21, 1824, p412, Medical Jurisprudence.

lost to history, especially given the inadequate methods of detection. Cases of arsenic poisoning thus set up the fundamental tension explored throughout the thesis. Given the tentative entrance of forensic medicine into jurisprudence in this period, and the limitations both of medicine and law in accommodating the practices of the other discipline, the conclusions one would initially draw from legal records as to the extent of poisoning as a social phenomenon are likely to be distinct underestimations. Limitations in detection, the presentation of evidence and variable standards of proof will have severely depressed the number of cases reaching trial and also decreased the chances of ‘guilty’ verdicts. The following chapters now turn to explore both similar and very different tensions between forensics and the law in cases of less well-known methods of poisoning.

APPENDIX 1

Arsenic Poisoning Cases 1806 - 1924

Date	Place	Accused	Victim(s)	Medium of Administration	Excuse for Obtaining Arsenic	What Happened to Victim(s)	Trial Verdict
1806	Fife	Margaret Cunningham	Husband- John Mason	Porridge	Not Known	Died	Guilty Executed
1811	Dumfries	Helen Kennedy	Master- Thomas Stothard	Porridge	Killing Rats	Died	Not Proven
1813	Kirkwall	Christain Sinclair	Niece- 8 mths	Porridge	Not Known	Died	Guilty Executed
1816	Edinburgh	Catherine Clerk	Mistress- Christain Ritchie	Tea	Killing Flies	Recovered	Guilty Banished
1818	Not Known	Robert Dingwall	Wife	Not Known	Not Known	Recovered	Not Guilty
1820	Argyle	Anne McEachern	Husband- Neil McKinnon	Porridge	Killing Rats	Recovered	Not Proven

APPENDIX 1 – (CONTINUED)

Date	Place	Accused	Victims(s)	Medium of Administration	Excuse for Obtaining Arsenic	What Happened to Victim(s)	Trial Verdict
1821	Aberdeen	George Thom	James Mitchell, Mary Mitchell, William Mitchell	Porridge and Salt	Killing Rats	James & Mary Mitchell Survived. William Mitchell Died	Guilty Executed
1822	Glasgow	Helen Rennie	Son	Direct	Not Known	Died	Not Guilty
1827	Dundee	Mary Elder or Smith	Servant – Margaret Warden	Medicine	Killing Rats	Died	Not Proven
1827	Arbroath	John Lovie	Servant – Margaret Warden	Porridge	Killing Rats	Died	Not Proven
1827	Arbroath	Margaret Wishart	Sister – Jean Wishart	Porridge	Not Known	Died	Guilty Executed
1831	Glasgow	Alexander Wingate	Mother & Sister	Porridge	Killing Rats	Both Died	Guilty Banished

APPENDIX 1 (CONTINUED)

Date	Place	Accused	Victim(s)	Medium of Administration	Excuse for Obtaining Arsenic	What Happened to Victim(s)	Trial Verdict
1832	Renfrew	George Frame	Mother & Sister	Porridge	Killing Rats	Both Died	Guilty Banished
1833	Fife	Andrew Williamson	Wife	Sugar	Killing Rats	Recovered	Not Proven
1835	Edinburgh	Elizabeth Banks	Husband-Peter Banks	Salts and water	Killing Rats	Recovered	Not Proven
1837	Carluke	Elizabeth Jeffray	Mrs Carl; Hugh Munro	Medicine Porridge	Killing Rats	Both Died	Guilty Executed
1839	Banff	James Mellis	Masters-Charles Hay & George Hay	Broth	Killing Rats	Both Recovered	Not Proven
1843	Glasgow	Mary MacFarlane or Taylor	Husband & Neighbour	Not Known	Not Known	Both Died	Technical Acquittal

APPENDIX 1 (CONTINUED)

Date	Place	Accused	Victim(s)	Medium of Administration	Excuse for Obtaining Arsenic	What Happened to Victim(s)	Trial Verdict
1844	Renfrew	Christina Gilmour	Husband- John Gilmour	Food and Drink	Killing Rats	Died	Not Proven
1845	Lockerbie	Janet Hope or Tedcastle	Son- George Tedcastle	Not known	Not Known	Died	Technical Acquittal
1845	Airdrie	James Mckerlie	George Stewart, John Stewart, Andrew Gold	Broth	Killing Rats	All Recovered	Guilty- Transported for Life
1846	Perth	Janet Campbell or McLellan	Husband- James McLellan	Tea and Scones	Killing Rats	Died	Guilty Executed
1849	Aberdeen	James Burnett	Wife- Margaret Burnett	Porridge	Killing Rats	Died	Guilty Executed
1849	Strathaven	Margaret Hamilton	Sister- Jane Hamilton	Not Known	Not Known	Died	Guilty Executed

APPENDIX 1 (CONTINUED)

Date	Place	Accused	Victim(s)	Medium of Administration	Excuse for Obtaining Arsenic	What Happened to Victim(s)	Trial Verdict
1850	Edinburgh	William Bennison	Wife- Jane Hamilton	Porridge	Killing Rats	Died	Guilty Executed
1852	Ross-shire	Sarah Fraser	Father- William Fraser	Porridge	Killing Rats	Died	Technical Acquittal
1852	Ross-shire	James Fraser	Father- William Fraser	Porridge	Killing Rats	Died	Technical Acquittal
1857	Glasgow	Madeleine Smith	Emile L'Angelier	Cocoa	Cosmetic Purposes & Killing Rats	Died	Not Proven
1859	Inverness	David Ross	Brother- Walter Ross	Porridge	Killing Rats	Died	Not Proven
1861	Stirling	Agnes Kirkwood	Master & his Son- Robert & William Kincaid	Porridge	Not Known	Both Recovered	Not Proven

APPENDIX 1 (CONTINUED)

Date	Place	Accused	Victim(s)	Medium of Administration	Excuse for Obtaining Arsenic	What Happened to Victim	Trial verdict
1869	Glasgow	Andrew Paterson	Mrs L.	Ointment	Medical purposes	Died	Guilty Culpable Homicide
1891	Kirremuir	John Webster	Wife	Food and Drink	Not Known	Died	Not Guilty
1896	Elgin	Ellen McLeod or Beaton	Master-Thomas Hay & 3 Farm Servants	Broth & Barley	Killing Rats-Kearney's Powders	All Recovered	Guilty – 15 months
1911	Dalkeith	John Hutchison	14 Dinner Guests	Coffee	Not Known	2 Dinner Guests Died	Accused Committed Suicide
1924	Edinburgh	William Laurie King	Mother & Father	Bread & Cheese	By Fraudulent Means	Mother Died	Not Proven

APPENDIX 2

Historical Tests Employed For Detecting Arsenic

A. Solid Arsenic¹⁷⁰

1. When thrown on to a red-hot iron will not flame, but rise entirely in thick white fumes.
2. Mixed with spirit of vitriol will give a light coloured precipitate which will harden into glittering crystals.
3. Mixed with spirits of salts will give a light coloured precipitate.
4. Mixed with syrup of violets will give a beautiful pale green tincture.
5. When exposed to a high temperature there will be dense white smoke and a peculiar odour of garlic.
6. A knife on which arsenic is burnt will become blackened.
7. On heating with muriatic acid, filtering and then boiling the filtered solutions with slips of copper ribbon the copper will be covered with a coating presenting all the external characteristics of a metallic oxide.
8. Heat white arsenic in a glass tube to approximately 400 degrees centigrade. The arsenious oxide vapour condenses on cooling to form octahedral or tetrahedral crystals of a remarkable lustre and brilliance.
9. When white arsenic is treated with a weak solution of ammonium sulphate in a watch glass and heated a rich yellow or orange-red film shall be left.

¹⁷⁰ Note

Tests 1-5 – Polson, C.J. & Tattersall., *Clinical Toxicology*, 2nd edn, Pitman, London, 1973, p205.

Tests 6, 15 – Marshal, John, “Remarks on Arsenic Considered as a Poison and a Medicine; to which are Added, Five Cases of Recovery from the Poisonous Effects of Arsenic”, *EMJ*, vol 13, 1817, p518.

Tests 7, 9 – Christison, Robert, “Processes for Detecting Poisons”, *Lancet*, vol 1, 1830-1831, p82.

Tests 8, 14, - Taylor, Alfred, Swaine, *On Poisons in Relation to Medical Jurisprudence and Medicine*, 2nd edn, Churchill, New Burlington Street, London, 1859, pp385-386.

Tests 10, 11, 13 are taken from Records of The Lord Advocate.

Test 12 – Christison, Robert, *A Treatise on Poisons in Relation to Medical Jurisprudence, Physiology and the Practice of Physic*, Adam Black & Longman, Rees, Orme, Brown & Green, London, 1829, p186.

- 10.** Heat white arsenic with a solution of hydrogen sulphide and hydrochloric acid and a yellow precipitate of arsenious sulphate will be formed.
- 11.** Heated on a platinum wire in a smokeless flame, powdered arsenic imports to the flame a pale blue colour, while it is volatized in white fumes.
- 12.** Mix arsenic with carbonaceous matter and then place this between two plates of polished copper. Bind this together with wire and then expose to red heat. The part of the copper, which is in contact with the arsenic, will be found to have received a permanent white stain.
- 13.** Arsenious oxide will dissolve in nitric acid and on addition of ammonium molybednate and warming a yellow precipitate will form.
- 14.** Reduction Test Mix arsenic or suspected powder with black flux or powdered charcoal, and put the mixture into a glass tube enclosed at one end, and coated with clay and sand, the other end being stopped with a plug of clay. Expose the tube to a red heat and if arsenic present it will be found in the metallic state encrusting the insides of the tube.
- 15.** A decoction of onions with solid arsenic mixed in will give a green colour with ammoniacal sulphate of copper and a yellow colour with ammoniacal nitrate of silver.

B. Liquid Tests to Determine Presence of Arsenic in Viscera¹⁷¹

1. When a minute quantity of alkaline hydro-sulphate is added to an acidified solution of suspect material an orange-yellow precipitate will be formed if arsenious oxide is present.
2. Scheele's Test. A beautiful green precipitate will be formed following the addition of the sulphate of copper to a solution containing the white oxide of arsenic to which potash has been added.
3. Ammoniacal nitrate of silver will give a lemon-yellow precipitate when added to any solution containing oxide of arsenic to which has been added: - carbonate of ammonia, or supercarbonate of potassium.
4. Sulphuret of potassium will give a bright yellow precipitate if arsenious oxide is present in solution.
5. Lime water will give a whitish precipitate when added to a solution containing oxide of arsenic.
6. Add a drop of water of carbonate of ammonia to the suspected fluid, then add nitrate of silver which will produce a yellow precipitate if arsenic oxide is present.
7. Sulphate of copper dissolved in distilled water will give a blue-green precipitate if arsenic oxide is present in the suspected fluid.

171 Note

Tests 1, 16 – Bostock, John, “Observations on the Different Methods Recommended for Detecting Minute Portions of Arsenic”, *EMJ*, vol 5, 1809, pp167-173.

Tests 2, 4, 10, 11, 12, – Taylor, Alfred Swaine, *On Poisons in Relation to Medical Jurisprudence and Medicine*, 2nd edn, John Churchill, New Burlington Street, London, 1859, p388, p390, p392, p397.

Tests 3, 5, 7, 9 – Christison, Robert, *A Treatise on Poisons in Relation to Medical Jurisprudence, Physiology and the Practice of Physic*, Adam Black and Longman, Rees, Orme & Green, London, 1829, pp187-188, p,191, p195.

Tests 6, 8, 13 are taken from Records of The Lord Advocate.

Test 14 - Campbell, W.A, “Some Landmarks in the History of Arsenic Testing”, *Chemistry in Britain*, vol 1, 1965, pp199-201.

Test 15 - Glaister, John, *Medical Jurisprudence and Toxicology*, 12th edn, E & S Livingstone Ltd, London, 1962, p679.

8. Improvement for Scheele's Test. Add a solution of potassium to one of sulphate of copper and then add to suspect fluid. If arsenic oxide present a blue precipitate will be obtained. If an arsenical solution is then added to this the precipitate will turn grass green or apple green.
9. Sulphuretted Hydrogen Test. Suspected fluid acidified with acetic acid and then subjected to a stream of sulphuretted hydrogen gas will give a rich orange precipitate if arsenious oxide is present. On reduction with black flux or charcoal this precipitate will yield patches of metallic crustations which on sublimation will yield little octahedral crystals of the oxide of arsenic.
10. Marsh Test – 1836. Prepare hydrogen from pure zinc and sulphuric acid in a solution containing the material to be tested. Burn the gas at a jet in the chemical apparatus, playing on cold porcelain or glass. If arsenic is present a metallic deposit will form on the cold porcelain or glass [Qualitative results only].
11. Marsh – Berzelius Test – 1837. Adaptation of equipment used in Marsh test by applying a flame to the existing tube. If arsenic present a metallic deposit forms which can be subjected to quantitative analysis.
12. Reinsch's Test – 1841. The liquid suspected to contain arsenic is boiled with about 1/3 of its volume of pure hydrochloric acid and a small piece of copper. If arsenic present in the liquid the copper acquires an iron-grey metallic coating from the deposit of arsenic metal.
13. Nitrate of silver, previously dissolved and applied to the suspected fluid itself will give a yellow precipitate if arsenic is present.
14. Gutzeit Test. The solution to be examined is placed in a wide-mouthed bottle to which is added 1 g of potassium iodide and 10 g of zinc. If arsenic is present a yellow stain is produced on mercuric chloride paper after one hour. By matching the depth of colour with standard stains the proportion of arsenic present may be detected.
15. Fleitman's Test. Zinc or aluminium reacting with sodium hydroxide when mixed with a solution containing arsenious compounds will result in the arsenic being reduced to arsine. The Gutzeit test can then be applied to detect arsenic.
16. Copper Test. When suspected liquid heated with muriatic acid and a slip of copper ribbon is added a coating will be found on the copper presenting the external characteristics of metallic arsenic, if arsenic is present.

APPENDIX 3

Post Mortem Appearances¹⁷²

1. Inflammation of the stomach, namely redness, softening or abrasion of its villous coat, and ulceration, sometimes penetrating the whole coat.
2. Gritty particles in the stomach due to conversion of arsenious oxide into the yellow sulphide.
3. Gritty particles in the intestines due to conversion of arsenious oxide into the yellow sulphide.
4. Coating of the stomach with mucous with streaks of blood.
5. Contents of stomach frequently a darkish-brown colour.
6. Inflammation of alimentary canal, viz. redness of the throat and gullet.
7. Redness and ulceration of duodenum and other parts of the intestinal canal.
8. Redness of inner coat of stomach.
9. Blackness of villous coat of stomach from effusion of altered blood into its texture.
10. Presence of a sanguinolent fluid or even blood in cavity of stomach.

¹⁷² **Note**

Aparances 1, 4, 5, 7, 12 – Taylor, Alfred, Swaine, *A Treatise on Poisons in Relation to Medical Jurisprudence and Medicine*, 2nd edn, John Churchill, New Burlington Street, London, 1859, p371-375.
Appearance 2 – Polson, C.J. & Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London, 1973, p202.

Aparances 3, 6, 8, 9, 10, 11, 13 – Christison, Robert, *A Treatise on Poison in Relation to Medical Jurisprudence, Physiology and the Practice of Physic*, Adam Black and Longman, Rees, Orme & Green, London, 1829, pp244-245, pp 249-251.

- 11.** Preservation of tissues.
- 12.** Villous coat of stomach easily detached from muscular.

Chapter 3

Vitriol Madness

3.1 Introduction

The characteristic feature of corrosive poisons is the power of chemical fixation and destruction of the surfaces with which the poison comes into contact.¹ There are at least thirteen corrosive acids known in science.² This chapter includes only four corrosive acids, those which were the most commonly known and the easiest to gain access to in the nineteenth and early twentieth centuries. The most common acid discussed in this chapter is sulphuric acid, also known as oil of vitriol. As sulphuric acid was extensively used for common purposes by manufacturers, craftsmen and artists this prevalence of usage for poisoning is not unexpected.

The signs and symptoms after swallowing a corrosive acid consist of burning, acute pain in the mouth, pharynx, oesophagus, and stomach, continuous retching and vomiting of shreds of blood-stained material, intense thirst, and often blockage of the air passages. Signs of corrosion may be noted of the mouth, lips or both. Consciousness is usually retained and death may result from shock, due to the extensive destruction of tissue, suffocation from swelling of the larynx, or perforation of the stomach. Of note is that although poisoning by corrosive acids would be accompanied by great thirst, a victim would be unable to obtain relief as he or she would be unable to swallow.³

Voluntary and accidental poisoning with corrosive acids did occur in the past.⁴ Few instances are on record, however, of a murderer having recourse to these poisons.

¹ Taylor, Alfred, S., *Medical Jurisprudence & Toxicology*, 12th edn, ed. Keith Simpson, Churchill, London, 1965, p235.

² Corrosive acids known include: Sulphuric acid; Nitric acid; Hydrochloric acid; Oxalic acid; Carbolic acid; Cresols; Lysol; Thymol; Pyrogallic acid; Naphthol; Chromic acid; Hydroquinone and Acetic acid.

³ Glaister, John., *Medical Jurisprudence & Toxicology*, 12th edition, E& S Livingstone Ltd, London, 1966, pp492-505.

⁴ Scott, David., *EMJ*, vol 24, 825, pp67-69, Case of Poisoning by Oxalic Acid, Successfully Treated; Sinclair, Martin., *EMJ*, vol 36, 1831, pp99-104, Case of Poisoning by Sulphuric Acid; Littlejohn,

Their intense and repellent taste and instantaneous corrosive action makes them much more difficult to administer than the innocuous arsenic, for instance. At first view one would therefore suppose it almost impossible to administer these corrosive poisons secretly.⁵ Different eras, however, give rise to different sorts of murders and according to Christison, sulphuric acid was used to murder infants on several occasions.⁶ Indeed, of the seven trials for murder and attempted murder with corrosive acids between 1800 and 1913 which are investigated herein, four of the victims were infants (57%).⁷ Although the figure of seven trials is extremely low compared to arsenic and represents just 11.5% of all poisoning cases it seems likely that the true figure for infants murdered in this way was much larger, especially in the cases of children of the working classes.⁸ Indeed, in the 1880s it was estimated that some 60% of all victims of murder by poison in England were less than a year old.⁹ In England between 1750 and 1914 corrosive acids accounted for 16% of all poisoning cases whilst at the Old Bailey this figure was 23% within the time period 1738-1878.¹⁰

Henry, D., *EMJ*, vol 7, 1861-1862, pp19-20, Case of Poisoning with Oxalic Acid in which Perforation of the Stomach Took Place.

⁵ Sulphuric acid or oil of vitriol (H_2SO_4); Nitric acid, aqua fortis or spirit of nitre (HNO_3); Oxalic acid ($COOH_2$) ; Carbolic acid or Phenol (C_6H_5OH).

⁶ Christison, R., *Treatise on Poisons*, 2nd edn, A & C Black, Edinburgh, 1836, p191 :- “A boy aged 2 $\frac{1}{2}$ years became drowsy and sick after drinking some “pop”, he said that his mother had given him. The fluid which the child was said to have drunk was submitted for analysis and shown to contain 94% sulphuric acid. Three hours after having drunk the substance the child died.”; “A one year old baby girl became seriously ill after her mother had administered some “medicine” to her. The child died several hours later and post-mortem analysis showed that sulphuric acid had been administered.”

⁷ $4/7 \times 100 = 57\%$. Given the fluid food diet of infants’ administration of such poisons would be easy.

⁸ I believe that often such murders would take place in order that the desperate parent or parents could collect the burial money to keep themselves alive. See also Tennyson’s *Maud*, (1855), Part I., I, Verse XII, lines 45-46, “When a Mammonite mother kills her babe for a burial fee, And Timour Mammon grins on a pile of children’s bones.”

⁹ Whorton, J.C., *The Arsenic Century: How Victorian Britain was Poisoned at Home, Work and Play*, Oxford University Press, p1.

¹⁰ See Watson, K., *Poisoned Lives: English Poisoners and their Victims*, Hambledon and London, London, 2004, p33; Forbes, T, R., *Surgeons at the Old Bailey*, Yale University Press, London, 1985, Table 8.

Poisoning with corrosive acids, in view of its suitability for criminal purposes and the extraordinary speed of fatality, never seems to have claimed the legal or scholarly attention it deserves. Murder by corrosive acids must undoubtedly have been aided by the lack of legislation controlling the sale of poisonous substances. Following the Arsenic Act of 1851, sixteen years passed before the Pharmacy and Poisons Act of 1868.¹¹ This Act, however, deemed only oxalic acid to be a poison within the meaning of the legislation. Forty years later, in the Pharmacy and Poisons Act of 1908, the list of controlled poisons had expanded to thirty-two, but still excluded the remaining corrosive acids.¹² In fact, it was not until the Pharmacy and Poisons Act of 1933 that the other corrosive acids were included in Part II of this Act, and some control exercised over their sale.¹³ Listed sellers, as well as registered pharmacists, however, could still sell poisons accounted for under Part II of the Act.¹⁴

Noteworthy, also, is that the provisions enumerated under the Act did not apply to a medicine containing a corrosive acid supplied by a duly qualified medical practitioner or dispensed by an authorised seller of poisonous chemicals. In relation to the seven cases discovered during the period in question for poisoning with corrosive acids, it would appear that the 1933 Act did have some impact on legally recognised cases of such poisoning, as I have discovered only one case which occurred after 1933.¹⁵

Similar to the recommendations of arsenic as a remedy which was observed in Chapter Two, the nineteenth century also witnessed the publication of material in mass circulation journalism, highlighting to the populace the beneficial uses of small

¹¹ The 1868 Act confined the sale of a range of poisons to qualified pharmacists.

¹² See Footnote 2.

¹³ Part II poisons contained those poisons in common use for domestic, agricultural or commercial purposes; eg: oxalic acid for household cleaning. For every sale of a Part II poison the following particulars had to be entered in a book – the quantity sold; the purpose for which it was stated to be required; date of purchase and name, address and occupation of purchaser.

¹⁴ This meant traders who had been authorised to sell such poisons by a local authority. Part I poisons could only be sold by registered pharmacists.

¹⁵ In all probability figures for suicides using corrosive acids fell due to the availability of less pain producing substances such as coal gas, barbiturates and synthetic narcotics.

quantities of corrosive acids as remedies.¹⁶ Corrosive acids were also extensively used in industry, and much promoted for use as domestic cleaning products. Carbolic acid was in common domestic use as a disinfectant, whilst nitric acid was used in the arts and industry, particularly for the manufacture of nitro-glycerine. Sulphuric acid was used in the arts and manufacturing industries and oxalic acid was used by shoemakers, bookbinders, brass polishers, straw-hat makers, and for domestic cleaning.¹⁷ Further, much press attention was given to corrosive acids because of trials for the new crime of disfiguring persons with acid.¹⁸

Given that corrosive acids were cheap to buy and that there was public awareness of their deleterious properties, it is of little surprise that they would be used as

¹⁶ Medical cases in which the use of sulphuric acid was indicated included treatment of syphilitic ulceration of the throat – Purdon, H.S., *BMJ*, vol 1, 1868, p447, A Case of Syphilitic Ulceration of the Throat Treated with Sulphurous Acid. Those in which the use of nitric acid was promoted included the removal of stones or gravel from the bladder – Copland, P., *EMJ*, vol 7, 1811, pp9-40, An Additional Account of the Lithontriptic Power of the Muriatic Acid; the treatment of sores in elephantiasis - Cook, E., *EMJ*, vol 13, 1811, pp8-19, Observations on the Effects of Nitric Acid in Elephantiasis. Carbolic acid was recommended for disinfecting and deodorising – Godfrey, B., *Half Yearly Abstract of the Medical Sciences*, vol XXXVII, 1863, pp330-331, On the Medical and Surgical Uses of Carbolic Acid; as an antiseptic following surgery in surgical dressings – Macphail, S.R., *EMJ*, vol XXVII, 1881, pp13-145, Carbolic Acid Poisoning (Surgical); as a local anaesthetic before surgery – M'Neill, R., *EMJ*, vol XXXI, 1886, pp1115-1119, The Analgesic Action of Carbolic Acid and the Cresol Group of Compounds.

It should be noted that, to prevent teeth being affected by the internal consumption of acids, doses were mostly sucked through a large goose-quill, and the mouth immediately rinsed with water.

¹⁷ The real value in oxalic acid, however, lay in its ability to remove stains. For example to take out spots of ink – “As soon as the accident happens, wet the place with juice of sorrel (oxalic acid) and hard white soap.” – Adams, S., *The Complete Servant*, Southover Press, Sussex, 1993, p107, reprinted from Adams S & Adams, S., *The Complete Servant*, London, 1825.

¹⁸ This crime originated in Glasgow during the industrial disputes of the 1820s, which took place in manufacturing districts between workmen and masters regarding the rate of wages. Following many instances of this malignant and cowardly mode of taking revenge, The Ellensborough Act against wounding and maiming introduced a clause which classed with other such crimes the offence in question and awarded the death penalty for such offences. See the case of Euphemia MacMillan in 1827, Hume, D., *Commentaries on the Law of Scotland Respecting Crimes*, p326. See also Record of the Lord Advocate for the cases of Anne Douglas – AD14/68/266; case of Theresa Kennedy – AD15/05/38; case of Susan Crawford – AD15/07/88.

instruments of murder.¹⁹ Cases of poisoning by corrosive acids are of particular medico-legal interest for two reasons. Firstly, because the use of a corroding fluid will usually result in burning and staining of the victim's clothing. The importance of this in the medico-legal investigations of the past was that there could be simple, positive detection of the use of a corrosive acid. Also it made available real evidence for production in court, in pointed contrast to poisoning by other substances. Secondly, and perhaps more importantly, is the principle that poisoning by corrosive acids highlights the theory advocated by forensic toxicologist Christison in the 1830s; that it was sometimes possible to infer poisoning absolutely from symptoms and morbid appearance alone.²⁰ The doctrine of the early nineteenth century that it was impossible, without chemical evidence, to form more than a presumptive opinion in favour of poisoning could, therefore, be overturned in the case of corrosive acids.²¹ These poisons thus highlight the development of the relationship between forensic medicine and the law from the perspective of a different set of engagements than those which appertained to the unreliable testing for arsenic.

3.2 Case History: An Overview

The seven cases in this chapter are powerful confirmation of Dr Christison's theory. Indeed, no cause other than the administration of some powerful corrosive could have produced the exact, sudden symptoms with fatal consequences.²² In all these cases

¹⁹ In 1800 the cost of a pound of oil of vitriol was listed as being 5d – Pierce, M., *London Medical Review*, vol 3, 1800, p108, Current Price of Drugs in the London Market.

²⁰ See Ryan, M., *London Medical and Surgical Journal*, vol 6, 1831, p406, Homicide by Poisoning; Christison, R., *EMJ*, vol 35, 1831, pp297-323, Cases and Observations in Medical Jurisprudence.

²¹ For the early doctrine see Dunglison, R., *Journal of the University of Virginia*, 1827, p33, Lectures on Medical Jurisprudence; Orfila, M.J.B., *Traité de Toxicologie*, 1814, p425; Paris, J.A., & Fonblanque, J.S.M., *EMJ*, vol 21, 1824, p27, Medical Jurisprudence. Compare with Christison, R., *EMJ*, vol 35, 1831, p297, Cases and Observations in Medical Jurisprudence – “This case is also important as illustrating the principle I have several times endeavoured to inculcate, - that it is sometimes possible to infer poisoning absolutely from symptoms and morbid appearances alone.” The case in question to which Christison is referring here is that of the murder of James Humphrey in Aberdeen by his wife Catherine in 1830.

²² Death was the end result for 6 (86%) of the victims in all seven cases.

death occurred within a period of twelve hours to two or three days.²³ It is of interest that, as with arsenic, the majority of the cases involving poisoning with corrosive acids were perpetrated by women (71%).²⁴ In contrast, however, to the arsenic poisoning cases, where only 53% of trials resulted in a guilty verdict, a verdict of guilty was returned in five (71%) of corrosive acid poisoning cases.²⁵ This figure also seems to lend support to Christison's theory that it was much easier to prove poisoning by corrosive acids beyond reasonable doubt, (due to staining on clothes and post-mortem appearances such as corrosion of the mouth and lips) than was ever possible with substances such as arsenic.

Out of seventeen guilty verdicts from the arsenic poisoning cases, 71% of the convicted were executed. However, for poisoning with corrosive acid the execution figure is only 40% of total guilty verdicts. The majority of trials (71%) for poisoning with acid occurred after 1851. This lower figure of execution is perhaps indicative, as noted in Chapter Two, of an emerging pattern in Victorian thinking and attitudes of greater leniency towards murder by poison, particularly, with reference to the murder of children with poison.²⁶ Thirty-four percent (34%) of arsenic poisoning cases resulted in 'Not Proven' verdicts. For poisoning with corrosive acids that figure is 29%, albeit it must be taken into account that this represents only two cases out of seven whereas for arsenic the figure is reached from eleven cases out of thirty-two.

3.3 Early Cases of Sulphuric Acid Poisoning

Poisoning is by its very nature a premeditated act. However, when considering any case, the circumstances of the life of the accused should also be taken into account. This consideration is particularly pertinent in the case of the trial of Barbara M.

I would contest this, saying that there can be an element of opportunity or spontaneity

²³ Death has even occurred within two hours – Christison, R., *EMJ*, vol 35, 1831, p405, Cases and Observations in Medical Jurisprudence.

²⁴ $5/7 \times 100 = 71\%$.

²⁵ $5/7 \times 100 = 71\%$.

²⁶ For more on the murder of children by poisoning during the Victorian era see the opium-poisoning chapter.

in 1807, when medical jurisprudence was just beginning as a science in this country.²⁷ At Edinburgh High Court in January 1808, Barbara Malcolm, an unmarried domestic servant, faced trial for the murder of her eighteenth month old daughter.²⁸ It was alleged that Barbara had administered oil of vitriol (or sulphuric acid) to her daughter on Tuesday 3rd December 1807, after which the child immediately began to cry, vomit and within a few hours died.²⁹ Post-mortem examination carried out by two surgeons on the 9th December 1807, by virtue of a warrant from the Sheriff of Edinburgh, pointed to poisoning with oil of vitriol from appearances alone.³⁰ No chemical analysis was carried out in this case.³¹

At trial the surgeons gave evidence that the child had died due to poisoning.³² Both surgeons believed this poison to be oil of vitriol. Clear and conclusive evidence was made further available by production of the child's clothes, which were corroded and the colour altered in parts.³³ A statement was made by the druggist, Mr Baird, that

²⁷ Andrew Duncan, senior, gave the first lectures delivered in the United Kingdom upon the subject of medical jurisprudence in the year 1801 at Edinburgh University and in 1806 his son, Andrew Duncan, junior, received the appointment of Professor of Medical Jurisprudence at Edinburgh University. It was not until 1839 that the first Professor of Medical Jurisprudence was appointed at Glasgow University, Robert Cowan.

²⁸ Justiciary Court Minute Book JC 8/5. Before and after the birth of her daughter, Barbara lived with her mistress, Mrs Hamilton, in Bristo Street, Edinburgh. Barbara's daughter, however, was given into the care of a Margaret Gordon of Lady Lawsons Wynd in Edinburgh. Barbara paid 20 shillings a quarter for this service. Barbara earned in the region of 5 guineas a year.

²⁹ Vomiting occurs at an early stage in cases of poisoning with oil of vitriol (sulphuric acid). Also with oil of vitriol poisoning children usually die at the end of a few hours – Polson, C.J., and Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London, 1973, p38.

³⁰ See note 21.

³¹ Appendix 9, appearances :- 2, 3, 4, 5, 6, 12, 15, 18, 20.

³² Proof of "corpus delicti" existed in that it was shown by evidence of morbid appearances that the child had not died from natural causes. This evidence was clear and further backed up by the evidence of corrosive burns on the child's clothing.

³³ The child's clothes consisted of 2 flannel petticoats, 2 shirts, a frock and a mutch (cap). There was also a child's handkerchief. No chemical tests had been applied to the corroded parts of the clothes – Justiciary Court Minute Book JC 8/5. The surgeons had only applied their tongues and tasted oil of vitriol and noted that there were stains of a dirty red-brown colour. Such stains are indicative of splashes of a corrosive acid or vomiting after swallowing a corrosive acid – Glaister, J., *Medical Jurisprudence and Toxicology*, 11th edn, E & S Livingstone Ltd, London, 1962 p39.

Barbara had purchased a penny worth of oil of vitriol from his chemist's shop on Monday 30th November 1807. This satisfied the first important principal circumstance with respect to establishing guilt in an alleged poisoning case.³⁴ Barbara finally confessed during the trial that she had both purchased oil of vitriol and then poured it down her daughter's throat, making it conclusive that she was guilty of the crime accused.³⁵ Accordingly, Barbara was found guilty by a majority of the jury and sentenced to death. She was executed on the 10th of February 1808.³⁶ This first case clearly illustrates Dr Christison's account of the possibility of the inference of poisoning from symptoms and morbid appearances alone.³⁷ It also highlights the facility of the acquisition of sulphuric acid and the potential for its use in the murder of infants.

The second case in this chapter also illustrates Christison's theory and indeed led to new tests for sulphuric acid poisoning.³⁸ The case is also notable for the minute circumstantial detail which connected the act of administration to the death in question. Mrs Humphrey, a butcher's wife in Aberdeen, was tried at Aberdeen High Court on September 10th 1830 for the murder of her husband with oil of vitriol

³⁴ This is proof of possession of poison. With regards to the other two principal circumstances, secret administration and motive, Barbara had every opportunity to administer the acid when she visited her daughter on the 8th December 1807 and spent time with her alone. Further, motive was present by the fact that the child was a huge financial burden to Barbara.

³⁵ The deed, said Barbara, had been performed of her own free will and she was now most sorry for her actions – Justiciary Court Records JC 26/338..

³⁶ An unmarried mother would have been placed in a desperate position during the nineteenth century and early twentieth centuries. Considering Barbara's situation and the course of action she took, it is perhaps strange that she did not get rid of the child when it was newly born and then claim that the child had been still-born. In particular, see Concealment of Pregnancy Act 1809, which reduced the crime of murder of a new-born baby to one of culpable homicide; although this Act would not have been of any relevance in this case as Barbara's child was born in 1805.

³⁷ See note 21.

³⁸ This second case led Dr Christison to introduce a new method for the detection of free sulphuric acid in stomach contents. This was because the previous test he had advocated did not give positive results for all corrosive acids - Christison, R., *EMJ*, vol 35, 1831, pp297-316, Cases and Observations in Medical Jurisprudence.

(sulphuric acid).³⁹ According to witnesses the prisoner and her husband had long lived on indifferent terms with each other.⁴⁰ They quarrelled often and were both frequently drunk.⁴¹ On Friday April 16th 1830 many people saw Mr Humphrey working at his butcher's market stall in Aberdeen. It was noted that he appeared to be in perfect health that day.⁴² Mr Humphrey returned home at eight o'clock that evening and held a drinking party with friends in the kitchen. In the next room his wife was drinking with female friends. When both husband and wife were somewhat drunk they quarrelled bitterly and exchanged blows.⁴³ Following this, the company left and Mr Humphrey retired to the servant's bed, located in a small room just off the kitchen, to sleep alone.⁴⁴ Mrs Humphrey went upstairs to sleep in the couple's bedroom. Within half an hour Mrs Humphrey woke the servant, Janet Petrie, wanting her to go and check on her master. On doing this Janet found Humphrey "crying out in pain, writhing from side to side and exclaiming that he was all burnt inside."⁴⁵ Medical assistance was sent for and the two attending doctors, Dr Jamieson and Dr Murray, noted symptoms of poisoning by a corrosive acid.⁴⁶ Despite extensive medical treatment James Humphrey died on Sunday evening, some forty-seven hours after the beginning of his illness.⁴⁷

³⁹ She was charged with perpetrating the crime by pouring the poison down her husband's throat whilst he was asleep. It was supposed that Mr Humphrey always slept with his mouth open – Record of the Lord Advocate AD14/30/157.

⁴⁰ Record of the Lord Advocate AD14/30/157.

⁴¹ During their quarrels blows as well as foul language were often exchanged and the prisoner on several of these occasions expressed a wish to various witnesses "that somebody would give her husband poison, so that she was kept clear of the matter" – Record of the Lord Advocate AD14/30/157.

⁴² Whilst working, however, he consumed several drams of whisky in the company of a number of acquaintances – Record of the Lord Advocate AD14/30/157.

⁴³ This was on account of the presence of one of Mrs Humphrey's acquaintances whose character was not to Mr Humphrey's liking – Record of the Lord Advocate AD14/30/157.

⁴⁴ The accused commonly slept upstairs, sharing the marital bed with the servant, following quarrels with her husband.

⁴⁵ Record of the Lord Advocate AD14/30/157.

⁴⁶ Burning in the throat, difficulty in breathing and swallowing, the tongue and inside of the mouth very white, the whole throat dark and inflamed and the pulse very feeble.

⁴⁷ Medical treatment tried included salt and water injections, a sinapism (mustard plaster) applied to the throat, administration of castor oil and magnesia and even the application of leeches in an attempt to help with breathing – Record of the Lord Advocate AD14/30/157.

Thirteen hours later a post-mortem examination was carried out by Drs Jamieson, Murray and Murray by direction of the Procurator Fiscal. From post-mortem appearances the doctors concluded that Humphrey had swallowed a highly corrosive substance, which was the cause of death, and that post-mortem appearances gave proof of "corpus delicti" in that Humphrey's death could not be attributed to natural disease.⁴⁸ It was the opinion of all three doctors that the substance swallowed had been oil of vitriol (sulphuric acid).⁴⁹ Despite the fact that no oil of vitriol was discovered in the stomach or intestines, chemical evidence was obtained by the application of various tests to the night-gown that Humphrey had been wearing and to his bedclothes.⁵⁰ Testimony, from Dr Christison, given during the trial of Mrs Humphrey corroborated this chemical evidence.⁵¹ In most trials for poisoning proof of the administration of poison by the accused is difficult to obtain. In this case, however, a chain of minute circumstances established it.

— In March 1830 servant Janet Petrie purchased three teaspoonfuls of vitriol for the accused.⁵² The phial in which the vitriol was kept stood at the kitchen window and was seen intact by the servant at five in the afternoon of Friday 16th April 1830. However, after Humphrey had been taken ill, the phial was found by one of the neighbours with only a few drops of vitriol left in it.⁵³ In addition, there was the discovery of an extra wineglass in the room in which the accused and her friends had

⁴⁸ Appendix 9, appearances -: 2, 5, 6, 7, 8, 9, 12, 13, 17.

⁴⁹ Sulphuric acid would have produced all the appearances noted by the three doctors. It is also the only strong corrosive acid which would have produced the brown mark observed on Humphrey's chin during post-mortem examination – See Christison, R., *Lancet*, vol 1, 1830-1831, pp133-135, Processes for Detecting Poisons; Sinclair, M., *EMJ*, vol 36, 1831, pp99-104, Cases of Poisoning by Sulphuric Acid.

⁵⁰ Appendix 5, tests -: 5, 6, 7, 8.

⁵¹ Appendix 5, tests -: 5, 6, 7, 8, 9.

⁵² The cost of the vitriol was a halfpenny and Mrs Humphrey had said that she required the vitriol for removing warts – Record of the Lord Advocate AD14/30/157.

⁵³ The phial containing the dregs of the acid, alleged to have been administered to Humphrey, was accidentally broken to pieces by the neighbour on discovery – Record of the Lord Advocate AD14/30/157.

been drinking. On tasting the contents of the glass a witness made the statement “that it was as if a lancet had been thrust on her tongue.”⁵⁴

During the trial of Catherine Humphrey the principal circumstances of proof of possession, opportunity for secret administration, and motive, were all proved. Motive was established by Mrs Humphrey’s frequent comments to friends and neighbours that she wished rid of her husband.⁵⁵ There was also the fact that Mrs Humphrey had attempted to hide her late husband’s night-gown and bed clothes from the police in an upstairs store room and was several times, whilst Humphrey was dying, overheard asking her husband to exculpate her.⁵⁶

During the trial the prosecution focused on the circumstantial evidence against the accused and the jury reached a unanimous verdict of ‘Guilty’. Catherine Humphrey was executed in pursuance of sentence on 8th October 1830 and just before her death made a full confession of her guilt.⁵⁷

It would seem that in the early nineteenth century obtaining evidence from corrosive acid poisoning cases could seldom be expected from chemical analysis of viscera alone.⁵⁸ On the other hand, it appears that conclusive evidence from symptoms and

⁵⁴ The contents of the wineglass were tasted following an incident on Sunday morning when a neighbour’s child, who was in this room, put said glass to his lips and then immediately began to scream violently – Record of the Lord Advocate AD14/30/157.

⁵⁵ “Damn the bugger, if any person would give him poison and keep my hands clear of it, I would be clear of him.”; “I would be well done to give him laudanum, to get rid of him.”; “I wish someone would give him poison so that I could keep well clear of the matter.” – Record of the Lord Advocate AD14/30/157.

⁵⁶ “Now lovey, clear me before these persons and say if I gave you it or not.”; “Oh James clear me afore Dr Jamieson for I am getting the blame of your death.”; “This is not my fault James, you have yourself to blame from your own doings.” – Record of the Lord Advocate AD14/30/157.

⁵⁷ Mrs Humphrey was the first woman hanged at Aberdeen since Elspet Reid in 1785, for theft by housebreaking. Catherine Humphrey’s body was given to Dr Charles Skene, Professor of Medicine at Marischal College in Aberdeen – Young, A.F., *The Encyclopaedia of Scottish Executions*, Eric Dobby Publishing Ltd, Kent, 1998, p105.

⁵⁸ This would be on account of the fluidity and easy miscibility of the poison with all other liquids in the body – Christison, R., *EMJ*, vol 35, 1831, pp297-322, Cases and Observations in Medical Jurisprudence.

morbid appearances could commonly be obtained. Unlike other poisons, the immediate burning and corrosive action of acids left such unmistakeable signs on the bodies of victims that doctors could be in no doubt as to the cause of poisoning.⁵⁹ Juries were arguably more likely to understand and convict on graphic descriptions of external morbid appearances rather than on evidence from complex chemical tests. The physically obvious morbidity of poisoning by acid also correlated with pre-Victorian and Victorian eras imaginings and glorifications of violent murder. Gaslit melodramas, puppets show, waxworks, peep shows, penny magazines and even Staffordshire figurines cast graphically depicted murder as popular entertainment. For this reason, perhaps murder by acid poisoning resonated more closely with juries' imaginings of 'murder' as a stereotype than did the discreet and doubtful appearances of arsenic poisoning for instance.⁶⁰

3.4 Mid-Century Lenience and Juridical Failures

Poisoning with oxalic acid, on account of its fitness for criminal purposes and its extraordinary speed of fatality, deserves more attention than it ever appears to have received.⁶¹ Although only two cases for poisoning with oxalic acid appear in the records it is likely that there would have been more incidents of deliberate and indeed accidental poisoning with this acid than are recorded since oxalic acid occurs in

⁵⁹ Watson, K., *Poisoned Lives: English Poisoners and their Victims*, Hambledon and London, London, 2004, pp10-11.

⁶⁰ See Altick, R.D., *Victorian Studies in Scarlet*, LM Dent & Sons Ltd, London, 1972, p10. That murder was popular entertainment in the past is comparable to the crime reporting during the 1950s, 1960s and 1970s by the media and in particular for Glasgow. During the 1970s Glasgow had a murder rate on a par with Northern Ireland and there were many television programmes and newspaper reports showing young gang members producing swords and hatchets for the camera. The distinguished political analyst Murray Ritchie wrote in the Glasgow Herald in an article around 1971 that "Glasgow is a violent, vandalised slum city." There have also been books such as Alexander McArthur's *No Mean City* and plays and films about Jimmy Boyle and others portraying the gang violence of the past in the Gorbals within Glasgow.

⁶¹ The first notice of the poisonous properties of oxalic acid seem to have been made by a Mr Royston in 1814 in the *London Medical Repository* – Royston, D., *London Medical Repository*, vol 1, 1814, p382, Poisoning Properties of Oxalic Acid. In this article Royston briefly related the history of a woman, who by mistake swallowed half an ounce of oxalic acid instead of sulphate of magnesia, and died in 40 minutes, after enduring the most horrible agonies.

nature in the leaves and young stalks of rhubarb (*rheum rhabonticum*), and also in wood sorrel (*rumex acetosa*) both of which were used in preparations designed to concentrate the acid for use as a tonic, diuretic or laxative.⁶²

On the 4th of December 1857 an unmarried mother, Margaret Macdonald, left the maternity hospital in Edinburgh where she had just given birth to a baby girl. On the 6th of December, accompanied by a neighbour, she went to the West Church poorhouse in Edinburgh to ask for help and support for herself and the baby. As they left, Margaret was observed to loiter behind her companion near a druggist's shop in Nicolson Street, and afterwards confessed that she had purchased there a penny worth of oxalic acid. The child was alive and apparently well at about nine o'clock that evening, but by ten o'clock was found dead in bed.⁶³

The body of the child was taken to the dead house of the local police station. There Professor Henry Littlejohn, forensic toxicologist, and a certain Dr Keiller carried out a post-mortem examination in consequence of a warrant issued by the local sheriff. Post-mortem examination of the appearance of the body revealed characteristics of the administration of some highly corrosive substance. The corrosive substance was thought to be oxalic acid.⁶⁴ Following removal of the viscera and chemical testing satisfactory evidence of the presence of this acid was obtained.⁶⁵ In addition to this a cloak in which the child had been wrapped was stained light yellow in several

⁶² Lust, J., *The Herb Book*, Bantam Books, Ltd, 1993, p334 & p359. Often in the past decoctions would be made from rhubarb or the root from wood sorrel by crushing and bruising the appropriate plant part in a pestle and mortar and then leaving them to soak overnight in boiling water. In the morning the mixture would be boiled and then strained to remove all the plant material. This would produce a standard decoction, which could be kept for 2-3 days, and taken neat- Hedley, C., *Herbal Remedies*, pp18-19. Rhubarb is still today used as an appetiser, astringent, purgative and tonic. It is also used in rhubarb crumble.

⁶³ See Littlejohn, H.D., *EMJ*, vol 7, 1861-1862, pp13-20, Case of Criminal Poisoning with Oxalic Acid, in which Perforation of the Stomach took place.

⁶⁴ Appendix 10, appearances -: 3, 7, 13, 14, 15, 18.

⁶⁵ Appendix 6, tests -: 3, 4, 8, 14, 15, 18. It was estimated from the amount of lead oxalate precipitated during chemical testing that nearly 4 grams of crystallised oxalic acid were present in the viscera.

places.⁶⁶ The stains on being cut out and washed in distilled water were proved by chemical testing to have been caused by oxalic acid.⁶⁷

The trial of Margaret Macdonald took place at Edinburgh High Court on the 23rd of March 1859, where medical evidence of the most conclusive kind was adduced for the Crown.⁶⁸ Proof of possession of poison was clear from the testimony of the druggist who had sold the oxalic acid to Margaret. Also Margaret had attempted to end her own life by swallowing a quantity of this poison.⁶⁹

In this case the accused had every opportunity to administer poison forcibly to her infant.⁷⁰ Further, the presence of motive was established by Margaret's parlous financial state in which she could barely afford to support herself let alone a child. Hence, the principal circumstances for establishing guilt in a poisoning case were all present.⁷¹ During the trial the defence argued that the prisoner was an ignorant, ill-educated woman and that she had given the poison to her child thinking it common salt.⁷² Margaret, however, admitted during the trial that she had given the child the

⁶⁶ Oxalic acid is a corrosive organic acid and does not corrode material in the same way as mineral acids such as sulphuric acid and nitric acid. These acids cause actual burning of material.

⁶⁷ Appendix 6, tests -: 4, 6, 7, 8.

⁶⁸ This gave proof of "corpus delicti", in that the child had died from administration of poison rather than natural disease.

⁶⁹ A stomach pump was, however, at once administered and suitable antidotes given rendering Margaret soon out of danger – Littlejohn, H.D., *EMJ*, vol 7, 1861-1862, pp13-20, Case of Criminal Poisoning with Oxalic Acid in which Perforation of the Stomach Took Place.

⁷⁰ "With infants and children of a tender age the most nauseous substances may be administered with facility by any one, and especially by the mother. Hence it is a rule in medical jurisprudence, that in such cases accident and suicide are out of the question, and that the poison must have been administered. This would be particularly the case for acids, the slightest touch of which would cause pain." – Littlejohn, H.D., *EMJ*, vol 7, 1861-1862, p17, Case of Criminal Poisoning with Oxalic Acid in which Perforation of the Stomach Took Place.

⁷¹ Proof of possession of poison, secret administration, motive.

⁷² This was because the prisoner was alleged to have seen common salt administered several times in the poor house to children to relieve them from pains in the bowels – Littlejohn, H.D., *EMJ*, vol 7, 1861-1862, p15, Case of Criminal Poisoning with Oxalic Acid in which Perforation of the Stomach Took Place.

“bonnet stuff.”⁷³ The evidence presented at trial conclusively pointed to the panel being guilty of the capital charge of murder and this was clearly suggested to the jury by the judge.⁷⁴ The jury after only fifteen minutes returned a unanimous verdict of guilty of culpable homicide.⁷⁵

This case is worthy of note on two points. Whilst the substance employed to cause death was comparatively rarely recorded as having been used as a poison in Scotland, there are much higher recorded figures in England.⁷⁶ Secondly the reduced verdict of culpable homicide was returned although there was clear intention to end life. Although fifty years had passed there seems little difference between the facts of this case and those of Barbara Malcolm earlier discussed in this chapter.⁷⁷ This verdict would, therefore, again attest to the argument that mid-late nineteenth century juries were possessed of an increased reluctance to sentence poisoners to death.⁷⁸

⁷³Littlejohn, H.D., *EMJ*, vol 7, 1861-1862, pp13-20, Case of Criminal Poisoning with Oxalic Acid in which Perforation in the Stomach Took Place. In the past oxalic acid was widely used when the straw hat industry flourished, between 1850 – 1910, to clean straw bonnets – Glaister, J., *Medical Jurisprudence and Toxicology*, 12th edn, E& S Livingstone Ltd, 1962, p64. “Hats were far more important than they are today. For hats in the past were not only invariably worn by all classes of society by day, but also with evening dress in restaurants, at the theatre and even by the female household servant just going for a walk at night on one of their days off.” – Ewing, E., *History of 19th and 20th Century Fashion*, Batsford Ltd, London, 1974, p13.

⁷⁴ Of note is that The Lord Justice-Clerk in charging the jury said, “This case is one of the most distressing, which has come under my cognizance for a long period. You have three courses, any one of which you can follow: absolve the prisoner, convict her of the crime of murder with which she was charged; or thirdly, it is in your power in cases such as these, if you see your way to it with safety, to return a verdict of culpable homicide.” – Littlehohn, H.D., *EMJ*, vol 7, 1861-1862, p16, Case of Criminal Poisoning with Oxalic Acid in which Perforation of the Stomach Took Place.

⁷⁵ The panel was sentenced to 15 months penal servitude.

⁷⁶ Between February 1845 and July 1846 there were 5 recorded trials at the Old Bailey for murder and attempted murder by oxalic acid. See Forbes, T.R., *Surgeons at the Old Bailey*, Yale University Press, London, 1985, p159.

⁷⁷ Justiciary Court Records JC26/338.

⁷⁸ In general see Opium Chapter and Conclusion.

The case of Mary Struth, tried at Edinburgh High Court on June 2nd 1862, for the administration of oxalic acid to her father, John Struth, presents singular features which differ from those adduced from the Macdonald trial. Firstly, it is the only case on record in which a charge of chronic poisoning of an adult with oxalic acid has been made in Scotland.⁷⁹ Secondly, it was of peculiar importance that the accused was not proved to have been in possession of the poison till the 11th January 1862, although the charge was that poisoning had begun on 5th December 1861. The serious nature of this defect in the evidence is apparent from the fact that symptoms proved to have occurred in Struth's case all throughout December 1861, which were consistent with poisoning by oxalic acid.

Mr Struth, a frail man of 75, lived with his daughter Mary, her illegitimate son, and a lodger, Elizabeth Maxwell, in Kincardine. On December 5th 1861 he became ill and ascribed his illness to a drink of dirty water his daughter had given him.⁸⁰ From that time onwards Struth was confined to bed and the accused was often seen by witnesses, giving him "medicine".⁸¹ Struth grew increasingly weak and appeared to be suffering from symptoms of corrosive acid poisoning.⁸² On the 11th January 1862 Mary purchased a pennyworth of salt of sorrel from grocer John Philip.⁸³ On returning home she pressed further "medicine" upon her father. She told him "it would do him good". Struth's condition immediately deteriorated and a Dr Crawford was sent for,

⁷⁹ A case of chronic poisoning stands in a different position to cases of "normal" poisoning. In such a case the quantity of poison given every time is very small until it gradually works into the system and begins to build up. In this way life the victim will suffer over a period of time and death will not be immediate.

⁸⁰ Record of the Lord Advocate AD14/62/258.

⁸¹ Witnesses included Elizabeth Maxwell – lodger; Mrs Shand – neighbour; Mrs Ainslie-neighbour :- Record of the Lord Advocate AD14/62/258.

⁸² Frequently during December 1861 Struth was unable to speak, complained of a sore breast, pain in his belly, burning in his throat and sickness. In addition, to the oxalic acid it was also alleged that Mary had given her father sugar of lead in porter during December. Sugar of lead was used in hair dyes and washes – Record of the Lord Advocate AD14/62/258.

⁸³ Record of the Lord Advocate AD14/62/258.

along with the inspector for the poor, Mr Steele, to jointly examine Struth.⁸⁴ An unfinished cup of “medicine” was noted to have been laid aside by the accused on the bedside cabinet in Struth’s room. On being asked what was in the cup, Mary flung the contents on the floor saying, “Do you think I would poison my father? It’s salt of sorrel, and I have it for cleaning the clothes”.⁸⁵ Dr Crawford administered magnesia as an antidote to salt of sorrel and had Struth moved, at his own request, to the house of a neighbour.⁸⁶ There Struth’s condition improved after the first week, but gradually relapsed until he died on January 26th 1862.

By virtue of a warrant from the Sheriff of Perthshire, a post-mortem examination of John Struth was carried out on January 28th 1862. This examination pointed to death by poisoning with oxalic acid.⁸⁷ Various organs were, therefore, removed and sent to Dr Maclagan, forensic toxicologist, in Edinburgh for chemical examination along with the cup which had contained the alleged salt of sorrel.⁸⁸ During the trial of Mary Struth, Dr Maclagan ascribed the death of her father to poisoning with salt of sorrel, providing proof of “corpus delicti”. His evidence corroborated that of the medical men who had performed the post-mortem. In addition there were the principal circumstances of proof of possession of poison by Mary, opportunities for secret administration and strong motive of financial gain.⁸⁹ To make out a case of chronic poisoning the Crown had to prove the continuous possession of oxalic acid by the

⁸⁴ Mr Steele had frequently visited Struth. This was, because, he was on the Poor Roll and in receipt of 2 shillings a week from this. During these visits Struth had frequently said to Mr Steele – “that vagabond (pointing to his daughter) has been giving me some stuff and wanting to kill me”. – Record of the Lord Advocate AD14/62/258.

⁸⁵ Record of the Lord Advocate AD14/62/258 When Mary was asked to get a spoon and lift the contents she scattered them further. Mr Steele, however, gathered up as much of the contents of the cup as he could.

⁸⁶ This was to the house of a Mrs Strang- Record of the Lord Advocate AD14/62/258.

⁸⁷ Appendix 10, appearances -: 3, 4, 5, 8, 10, 18.

⁸⁸ The chemical tests performed by Dr. Maclagan proved the presence of salt of sorrel in the viscera and the cup – Appendix 6, tests -: 1, 3, 5, 6, 7, 10, 11.

⁸⁹ Evidence given by the secretary of the Kilbagie Death Fund Society proved that the deceased was a contributor to its fund and that £6 would be payable on death to Mary. Mary had also made frequent enquiries to ensure that she would be the recipient of any money due after the death of her father.

accused.⁹⁰ This, however, they failed to do and the jury by a majority found the charges against Mary ‘Not Proven’.⁹¹

On comparison of the features of this case with the other case of poisoning with oxalic acid, it seems clear that, while it would not have been safe for the jury (on the evidence before them) to convict the prisoner of murder, the conclusion at which science must arrive is that the death of Struth was caused by the administration of oxalic acid.⁹² It would seem that in this case a review of the evidence is convincing enough to show that the charge of administration with intent to destroy life or cause grievously bodily harm was proved in regard to the administration of salt of sorrel on the 11th January. It is possible; therefore, that justice was defeated in this poisoning case due to the serious defect in the nature of the evidence or the mistaken choice of wording in the charge.

⁹⁰ This is because three occasions were specified on the indictment, - viz., 5th or 6th December 1861, 29th December 1861, and 11th January 1862, on the first and last of which salt of sorrel was alleged to have been administered; and on the second of which sugar of lead was alleged to have been administered- Cowan, H., *EMJ*, vol 8, 1862-1863, pp93-101, Report of the Trial of Mary Struth for Poisoning with Oxalic Acid, with Remarks.

⁹¹ The Lord Justice-General, in charging the jury, said that this was undoubtedly a case of great suspicion, but that the jury must consider attentively the evidence put before them. Having explained the nature of the indictment, he pointed out that there had been no poison traced into the possession of the prisoner prior to 29th December, and that the lead found in the body of the deceased was in such small quantity that it might have come there naturally, and that could not be said to be the cause of death. That no salt of sorrel was traced to the prisoner till the 11th of January, and that it was in evidence that even if they came to the conclusion that salt of sorrel had been administered as charged, that day, the death of the deceased should have resulted more quickly if caused by that dose. That to make out a case of chronic poisoning the Crown should have proved the continuous possession of the poison by the prisoner – Cowan, H., *EMJ*, vol 8, 1862-1863, p101, Report of the Trial of Mary Struth for Poisoning with Oxalic Acid, with Remarks.

⁹² This case can also be considered in a modern context given the present public interest in herbal remedies with there being little awareness of the potential harm from some herbal preparations. For example, although the rootstock of rhubarb can be used as an appetiser, astringent, purgative and tonic the leaves contain enough oxalic acid to cause poisoning. In addition, although wood sorrel is used as an analgesic, diuretic and in salads, the leaves contain oxalic acid and excessive amounts will result in poisoning. See also note 62.

A case of poisoning by oil of vitriol occurred in February 1869 in Dundee.⁹³ This is the first case discovered where an adult male administered a corrosive acid to an infant.⁹⁴ Stewart Ogilvie, father of the child, was alleged to have poured acid down the child's throat, during a meeting with his estranged lover, Mary Luman and mother of their son, David.⁹⁵ The three month-old male infant died from exhaustion and starvation twenty-four days later. Initial medical examination of the living child, by a Dr Smith, revealed characteristics of burning by a corrosive substance.⁹⁶ In addition, the child's gown and shawl both had burn marks and a strong smell of vitriol, (in cases of poisoning with vitriol children usually die after a few hours).⁹⁷ That the infant lingered for twenty-four days is indicative that the acid had been diluted.⁹⁸ It is likely that Ogilvie feared that the acid would be too strong for the child to swallow if undiluted.

Under instruction from the Procurator Fiscal a post-mortem examination was carried out on the body of the child, on March 24th 1869.⁹⁹ The appearances the body presented were such that the opinion was given that death had resulted from poisoning by oil of vitriol.¹⁰⁰ Further to this was the chemical analysis of the child's shawl and dress, which yielded positive results for the presence of oil of vitriol.¹⁰¹ At trial in Dundee High Court on September 6th 1869, the medical evidence was conclusive that the child had died from poisoning with oil of vitriol. Proof of "corpus delicti" was established, but whilst the principal circumstances for establishing guilt of secret administration and motive were proved, there was no proof of possession of poison by

⁹³ Record of the Lord Advocate AD14/69/130.

⁹⁴ Only two of the seven cases in this chapter (29%) involved administration of corrosive acid to a child by a male.

⁹⁵ The couple had met in Dundee in order that Ogilvie could make arrangements for financing his child. During the discussions Ogilvie sent Mary to a local confectioner to purchase sweets and insisted she left the child alone with him – Record of the Lord Advocate AD14/69/130.

⁹⁶ Foetid breath, vomiting of a tough mucous, small, irregular and weak pulse and great thirst.

⁹⁷ Christison, R., *Treatise on Poisons*, 2nd edn, A & C Black, Edinburgh, 1836, pp137-138.

⁹⁸ Diluted acid would cause a more prolonged illness which would last several weeks – Polson, C.J., and Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London, 1973, p38.

⁹⁹ This was by a Dr Pirie and a Dr Mackie.

¹⁰⁰ Appendix 9, appearances -; 1, 4, 6, 7, 9, 10.

¹⁰¹ Appendix 5, tests -: 5, 6, 7, 8, 9.

Ogilvie. Motive, however, was particularly strong with regard to Ogilvie's reluctance to pay money for the upkeep of the child, and his overheard threats to Mary when she accused him of poisoning her child.¹⁰²

Ogilvie pleaded not guilty at trial. The jury, however, by a majority found him guilty of administering poison to his child with intent to destroy life. Surprisingly, however, the jury recommended leniency on account of his youth and previous good character.¹⁰³ In comparing this case with the case of Barbara Malcolm this verdict seems hardly just.¹⁰⁴ It seems likely, therefore, that once again this case suggests the reluctance of those living in the mid Victorian era to sentence poisoners to death. Further, given that juries of this period were all male, it is possible that the panel was looked upon with more sympathy than a female accused.

3.5 Late Cases: Nitre and Carbolic Acid

Poisoning by nitric acid or by nitre, the organic salt of the acid, appears to have been less common than the use of other acids.¹⁰⁵ The taste and the rapidity of the action of nitre are naturally serious obstacles to its use by poisoners, and, thus children are the most likely victims. However, given that nitre was even less restricted by the course of legal reform on the sale of potentially lethal substances, the lack of cases of poisoning using these substances is surprising.¹⁰⁶ Nitre was sold in every grocer's shop, with its purchase attracting no attention.¹⁰⁷

¹⁰² Ogilvie was heard by passer by Henry Hunter to say to Mary that "if she did not hold her tongue, he would do something to her". – Record of the Lord Advocate AD14/69/130.

¹⁰³ Ogilvie was a 25-year-old flesher. He was sentenced to only 8 years imprisonment - Record of the Lord Advocate AD14/69/130.

¹⁰⁴ Barbara Malcolm was executed in 1808, for the murder of her child with oil of vitriol.

¹⁰⁵ Tatra, the renowned French forensic toxicologist, whose monograph remains a principal source of information, was able to collect a series of only 56 cases - Tatra, A.E., *EMJ*, vol 9, 1813, p23, *Traité de L'empoisonnement Par L'acide Nitrique*.

¹⁰⁶ Nitre is also known as potassium nitrate or saltpetre a combination of nitric acid with potash. It was not even controlled by the Pharmacy and Poisons Act of 1933.

¹⁰⁷ Nitre was used as a medical inhalant, diuretic, purgative and preservative.

The case of Elizabeth Walker in 1884 is thus singular in that it represents the only case of criminal administration of nitre recorded in Scotland. On a Friday evening in November 1884 Elizabeth Walker and her landlord called at the home of Dr Henry Littlejohn in Edinburgh and requested him to issue a Certificate to the Registrar as to the death of Elizabeth's child.¹⁰⁸ The parties were evidently in poor circumstances and Littlejohn agreed to issue a certificate, if he were allowed to make a post-mortem examination. This, the mother refused to allow and according to Littlejohn "her manner in doing so was so peremptory and peculiar that I determined to mention the case to the police that evening, and request them, on behalf of the authorities, to make some inquiry into the circumstances of the case." Littlejohn accordingly spoke with the local police sergeant to express his concerns.¹⁰⁹

Next morning the Procurator Fiscal, on being made acquainted with the circumstances of the case, at once gave instructions for a post-mortem examination of the child to be carried out by Dr Harvey Littlejohn and the parochial medical officer of the district where death had occurred.¹¹⁰ From examination, both doctors were of the opinion that the death of three year old William Walker had been caused by the action of some irritant substance on the stomach and bowels.¹¹¹ The viscera were, therefore, removed and sent to Dr Maclagan, forensic toxicologist in Edinburgh, for chemical testing along with a pair of child's boots, which appeared to have minute crystals on the

¹⁰⁸ Dr Littlejohn was told that death had occurred somewhat suddenly that afternoon – Littlejohn, H.D., *EMJ*, vol XXXI, 1885, p102, *The Practice of Medical Jurisprudence: With Hints as to the Conduct of Medical Practitioners in Cases of Suspected Poisoning*.

¹⁰⁹ As Littlejohn was speaking to the sergeant two women made their appearance and detailed what seemed to them suspicious facts as to the sudden death of a child in their immediate neighbourhood. The following statement was obtained from the witnesses, viz.: - That the child was illegitimate, and that the mother had been heard to complain of the burden of its maintenance, and to speak harshly to it; that the child was a remarkably fine healthy boy, and a favourite among the neighbours. That that afternoon it had been taken out by its mother and brought back in the course of half an hour ill and vomiting, and shortly afterwards they were surprised to hear of its death - Littlejohn, H.D., *EMJ*, vol XXXI, 1885, p102, *The Practice of Medical Jurisprudence: With Hints as to the Conduct of Medical Practitioners in Cases of Suspected Poisoning*.

¹¹⁰ The mother had also requested the parochial medical officer of the district to certify the death of her child after the refusal of Dr Harvey Littlejohn to do so. The parochial medical officer had also refused to comply with this request – *Ibid*, p103.

¹¹¹ Appendix 11, appearances :- 3, 4, 7, 10.

toe-parts, and other articles of child's clothing recovered from the house.¹¹² From their chemical reports Drs Littlejohn, Maclagan and the parochial medical officer concluded that the child must have swallowed a large dose of nitre, which led to his death.¹¹³ Elizabeth Walker was, therefore, held to await trial on the capital charge.

During the trial, evidence adduced showed that the child had been taken out by his mother in good health at 5.35pm on the Friday in question, and brought back within the course of half an hour, cold, sick and vomiting. Without doubt, proof of "corpus delicti" existed in that the medical evidence was clear and conclusive that the child had died from poison and not natural disease. Further, the principal circumstance of opportunity for secret administration was proved at trial, as was motive via the testimony of many neighbours who spoke of Elizabeth Walker being unwilling and financially unable to support her illegitimate son.¹¹⁴

With the strong medical evidence, it seemed during trial that the prisoner was guilty. However, on one point the evidence for the Crown was deficient, in that there was no proof of purchase of poison.¹¹⁵ This deficiency in the Crown evidence was strongly

¹¹² Appendix 7, tests :- 1,2,5,6,7,12.

¹¹³ "That from our whole experiments we can come to no other conclusion than that this child must have swallowed a very large dose of nitre, which had not only impregnated the blood and all the organs, but had been discharged in considerable quantity in the matters vomited and passed by stool." - Littlejohn, H.D., *EMJ*, vol XXXI, 1885, p106, *The Practice of Medical Jurisprudence: With Hints as to the Conduct of Medical Practitioners in Cases of Suspected Poisoning*.

¹¹⁴ There were several public wells in the neighbourhood and testimony was given by two independent witnesses that Elizabeth Walker was seen taking water from one of these between 5.35 p.m. and 6.00 p.m. Further the child was illegitimate and the mother had been heard to complain frequently of the burden of maintenance and speak harshly of it.

¹¹⁵ The accused lived in a crowded neighbourhood, and on the local tradesmen being interrogated as to their sales on the day in question several remembered selling quantities of nitre, but when Elizabeth Walker was placed amongst other prisoners they failed to recognise her. At last one grocer spoke distinctly to selling a small package of the substance to a female as he was engaged in transacting some business with a commercial traveller, and he identified a shawl which the accused had borrowed from a neighbour and had on that evening, but on allowing the woman to mix freely with other females the witness could not positively say she was the purchaser – Littlejohn, H.D., *EMJ*, vol XXXI, 1885, p107, *The Practice of Medical Jurisprudence: With Hints as to the Conduct of Medical Practitioners in Cases of Suspected Poisoning*.

commented on by the defence, and it was also urged on behalf of the accused that the poisoning might have been accidental.¹¹⁶ Such a line of defence seems highly improbable. An important link in the chain of evidence was, however, missing, and the jury returned a unanimous verdict of ‘Not Proven’. Although this verdict affirmed the strong suspicion attached to the prisoner and justified the action of the Crown in bringing her to trial, the jury very properly gave the accused the benefit of the doubt due to the missing link in the evidence.

This case highlights, since no record of purchase could be recalled, that traffic in this particular poison attracted little attention in the past.¹¹⁷ A wider conclusion is also prompted by this case. It seems possible that juries of the past were likely to form a presumptive opinion in favour of poisoning only if there was absolute proof, at trial, of purchase and possession of poison by an accused.¹¹⁸ This further highlights juries’ distrust of medical evidence in the nineteenth and early twentieth centuries¹¹⁹ Proof of the “corpus delicti” appeared to rest more with the other circumstances of the case than with medical evidence. It would seem, therefore, as if during the late 19th and early 20th centuries there was waning trust in the study of forensic toxicology by the

¹¹⁶ The defence argued that such a common substance as nitre lying exposed in shops might be spilt on the floor and thus swept into the street. Should the child have been left alone, its attention might have been attracted to the white deposit in the gutter. On tasting this, the child, pleased with its coolness, would be inclined to take more, and thus it was possible a heedless child might take a poisonous quantity. Or again, the child, if left unprotected by its mother, might have the poison administered to it by some unknown party - Littlejohn, H.D., *EMJ*, vol XXXI, 1885, p106, *The Practice of Medical Jurisprudence: With Hints as to the Conduct of Medical Practitioners in Cases of Suspected Poisoning*.

¹¹⁷ The cost in 1884 was roughly £4.14s.6d. for 1cwt of nitre (1lb of nitre would have cost approximately 10d.) – Pierce, M., *London Medical Review and Magazine*, vol 4, No. X11, 1884, *Current Price of Drugs in the London Market*.

¹¹⁸ The first principal circumstance in which reliance can be placed for establishing guilt or innocence is possession of poison by an accused. See Arsenic chapter. See also trial of Stewart Ogilvie for murder with sulphuric acid – Record of the Lord Advocate AD/14/69/130. Also trial of Agnes Kirkwood for attempted murder with arsenic - Record of the Lord Advocate AD14/61/79.

¹¹⁹ See Advocate, H.M. v. Elder (or Smith) 1827 Syme 71, per the Lord Advocate at p128: “Amongst science there were uncertainties there were blunders, and it was the pride of one age to tear up theories to be trampled down and triumphed over by the next.” See also Hamlin, C., *Social Stud. Sci*, vol 13, 1986, pp485-513, *Scientific Methods and Expert Witnessing, Victorian Perspectives on a Modern Problem*.

populace.¹²⁰ As Burney comments, the inability of toxicology to produce either the concrete proofs or the infallible verdicts that the Victorian public desired led to criticism of the “inflated ambitions of medical witnesses”¹²¹,

The seventh case involved poisoning by carbolic acid (phenol) which was obtainable as, a common disinfectant.¹²² Given, its very distinctive and repellent smell however, it seems an unlikely choice for poisoning. It was not indeed, until the Poisons and Pharmacy Act of 1908 that it was even recognised as a poison and strict controls on its use and sale were not put in place until the Pharmacy and Poisons Act of 1933. It was often thus a poison of choice in suicides. In the case at hand, Daniel Lipp, a forty-one year old mill furnisher’s porter in Dundee, secretly planned to murder his wife

¹²⁰ This is in sharp interest to the interest of the populace in medicines and especially quack medicines. In particular during the reign of Queen Victoria chemists’ and druggists shops were crammed with a profusion of proprietary pills, powders and potions to meet the need of a demanding population. See Porter, R., *Quakes, Fakers and Charlatans in English Medicine*, Tempus Publishing Ltd, Gloucestershire, 2000.

¹²¹ Burney, I., *Poison, Detection and the Victorian Imagination*, Manchester University Press, 2006, p133.

¹²² Phenol (monohydroxybenzene) is commonly known as carbolic acid. It occurs as colourless needles, which have a low melting point (42.5-43 degrees Celsius). However, aqueous solutions rather than pure phenol are usually the cause of poisoning. Phenol was used extensively in the past as an antiseptic agent and gave rise to many cases of medicinal, accidental, and suicidal poisonings – Dubois, K.P., and Geiling, E.M.K., *Textbook of Toxicology*, Oxford University Press, New York, 1959, pp119-120. Today carbolic acid (phenol) has limited use, because less toxic compounds have replaced it. It is still, however, used cosmetically in skin-peeling agents – Olsen, K.R., *Poisoning and Drug Overdose*, Appleton & Lang, Stamford, Connecticut, 1999, p255. Of interest is that carbolic soap, which used to be a standard presence in every home and school in this country, can still be purchased. On Wednesday, 14th August 2002 Judge Keith Simpson told Maidstone Crown Court that a dose of carbolic might be the best way to clean up bad language. He spoke out while sentencing a man who shouted racist abuse and threats in front of children in a Kent shop. Peter Moore, 49, of Beckenham, Kent, was ordered to do 100 hours of community service and to attend anger management classes after swearing at staff at a furniture store in Chatham in Kent. The judge told Maidstone Crown Court: “There was a time when young children who picked up bad language would have their mouths washed out with carbolic soap. I don’t know if carbolic soap is still available but it might be no bad thing if the courts were empowered to take similar action.” – Baker, M., *BBC News Online*, Wednesday, 14th August 2002, Wash out Foul Mouths says Judge.

Jane.¹²³ On December 26th 1910 Jane Lipp took a tablespoon of her prescribed medicine.¹²⁴ She immediately smelled carbolic acid and felt a burning sensation in her mouth and throat. Jane went at once to see her local doctor, Dr Macvicar, who washed out her stomach, preserved the contents and called the local constable due to his suspicions of poisoning.

On being arrested and charged with attempting to poison his wife Lipp threatened to poison himself. In addition, a note was found in his possession saying, “Good Bye all, God Bless, you have been the means of this.”¹²⁵ Daniel Lipp was tried at Dundee High Court on April 4th 1911 for attempting to murder his wife. Chemical testimony, given by the city analyst for Dundee, Dr MacDougal, showed that 0.07 grams of carbolic acid had been extracted from the medicine bottle of which Mrs Lipp had partaken. Evidence from Dr Macvicar during the trial attested that Mrs Lipp had taken carbolic acid. That a bottle of carbolic acid had been purchased in October 1910 by Mr Lipp (and still lay in the house nearly full) was never in doubt during the trial, as was the fact that Lipp had insured his wife’s life for £12-7s-3d.¹²⁶ Further, on Friday 23rd December 1910, Lipp had enquired of his wife how much carbolic acid it would take “to do away with one.”¹²⁷ Lipp pleaded not guilty to the charge against him, and, during his trial the defence argued that he had meant to poison himself due to depression. The jury, however, unanimously found him guilty and he was sentenced to twelve calendar months in prison¹²⁸

In a further case, admittedly external to the given time period, in August 1937 James Grant was charged at Fort William Sheriff Court with the murder of one year old

¹²³ Record of the Lord Advocate AD15/11/149.

¹²⁴ Jane Lipp had been regularly taking *mistura ergotae*, prescribed by Professor John Alexander Konoch of Dundee University College for inflammation of the womb. This consisted of liquid extract of ergot, dilute sulphuric acid and infusion of roses – Record of the Lord Advocate AD15/11/149.

¹²⁵ Record of the Lord Advocate AD15/11/149.

¹²⁶ This had been with the Prudential Assurance Company only 30 days before Jane Lipp swallowed the carbolic acid put in her prescribed medicine – Record of the Lord Advocate AD15/11/149.

¹²⁷ Record of the Lord Advocate AD15/11/149.

¹²⁸ Ibid. There was proof of possession of poison, opportunity for secret administration and motive present by Lipp’s hope for financial gain by insuring his wife’s life. All principal circumstances were, therefore, present on which reliance can be placed for establishing guilt or innocence.

Mary Williamson, the daughter of his wife Margaret from a previous relationship.¹²⁹ It was alleged that Grant had put ear lotion containing carbolic acid, into a bottle of milk to be fed to the child with the intent to do it grievous bodily harm.¹³⁰ Carbolic acid was frequently used as a solution in glycerine for eye drops or as a mouth wash in the past. Grant was found guilty of murder, but on appeal his conviction was overturned due to an issue that had arisen in relation to admissibility of evidence during his trial.

3.6 Conclusions

It seems possible that, as with arsenic, corrosive acids were more extensively employed for murder during the 1800s and early 1900s than the records show. This conclusion is especially persuasive given the prevalence of the use of such substances for the murder of infants. Indeed, it would appear to have required no more than common prudence and tolerable competence to poison a child successfully.¹³¹ The means were easily available and the motives for wishing rid of children, especially those born out of wedlock were substantial. Furthermore, the prospect of detection of poisoning was sufficiently remote to make the risk worth taking for many. With many of the poor dying far from the reach of the medical profession, it would only have been if doubt were already entertained that official questions were likely to have been asked.

These conclusions may be supplemented by consideration of the relationship between forensic medicine and the law suggested by these cases. Unlike in the cases involving arsenic poisoning, the establishment of positive proof of death by corrosive acid was not difficult. Thus, the higher rates of ‘guilty’ verdicts are not surprising. However, the existence of ‘not proven’ and ‘culpable homicide’ verdicts attests to a continued unwillingness to convict for murder based solely on the facts of medical testimony. When circumstances such as proof of purchase could not be established, juries were unwilling to pronounce guilty verdicts. Treating medical testimony as only

¹²⁹ Grant v. H.M. Advocate. 1938 J.C.7; Justiciary Court Records 1938 JC7.

¹³⁰ Grant v. H.M. Advocate 1938 J.C.7, p8.

¹³¹ Massie, A., *Ill Met by Gaslight*, p58, Futura Publications, London, 1987.

corroborative of the circumstantial facts of the case is consonant with the hesitant relationship between juries and forensic science which has been noted throughout.

APPENDIX 4

CORROSIVE ACID POISONING CASES 1807-1937

Date	Place	Accused	Victim(s)	Medium of Administration	Excuse for Obtaining Corrosive Acid	What Happened to Victim(s)	Trial Verdict
1807	Edinburgh	Barbara Malcolm	Daughter- Margaret Sutherland- 17 mths	Direct	None Given	Died	Guilty Executed
1830	Aberdeen	Catherine Humphrey	Husband- James Humphrey	Direct	For Warts	Died	Guilty Executed
1857	Edinburgh	Margaret MacDonald	Own child- 2 days old	Direct	None Given	Died	Guilty Culpable Homicide
1861	Kincardine	Mary Struth	Father- John Struth	In Water and Medicine	None Given	Died	Not Proven
1869	Dundee	Stewart Ogilvie	Son- David Luman 3 yrs	In Water	None Given	Died	Guilty- 8 years
1884	Edinburgh	Elizabeth Walker	Son - William Walker - 3yrs	Direct	None Given	Died	Not Proven
1910	Dundee	Daniel Lipp	Wife - Jane Lipp	In Medicine	None Given	Survived	Guilty - 12 months
1937	Fort William	James Grant	Daughter- 1 yr	In Milk	None Given	Died	Guilty- overturned on Appeal

APPENDIX 5

Historical Tests Employed For Detecting Sulphuric Acid¹³²

1. Will redden litmus paper or if litmus paper not available use unsized paper dyed in a decoction of red cabbage.
2. Will rapidly corrode animal substances when concentrated.
3. Taste Test. Dilute suspect fluid and taste. If sulphuric acid (or other mineral acid) is present there will be an acute sense of burning
4. Boil suspect matters for a few minutes, and after filtration add subcarbonate of lime. Agitate mixture then add solution of nitrate of baryta and a little nitric acid. If a heavy white precipitate falls down it can be nothing else than sulphate of baryta, because no acid but the sulphuric forms with the barytic salts a white precipitate insoluble in nitric acid.
5. To above precipitate of sulphate of baryta after filtering and washing add 2 grains of dry charcoal powder and heat for 2 minutes in a covered platinum spoon. A portion of sulphate of barium will be converted to sulphured barium. To prove put powder into a test tube add water and a little hydrochloric acid and hold within the tube white paper moistened with nitrate of lead. Sulphurated hydrogen gas will evolve which will darken the paper and betray itself by its odour
6. To a suspect portion of bed covers, clothing etc., add compounds of barytes which will give precipitates insoluble in nitric or muriatic acid which when heated along with charcoal and then placed in contact with water and muriatic acid will give sulphurated hydrogen gas if sulphuric acid is present.
7. Any stains on material will appear yellowish brown.
8. Drop on fragments of material already suspected to be corroded with sulphuric acid 2 drops of concentrated sulphuric acid and compare.

¹³² Note

Tests 1, 2, 3, 4, 5, 6 – Christison, Robert, “Processes for Detecting Poisons”, *Lancet*, vol 1, 1830-1831, p133.

Tests 7, 9, 10 – Christison, Robert, “Cases and Observations in Medical Jurisprudence”, *EMJ*, vol 35, 1831, p313.

Tests 8, 11, 12, 13, 14, 15 are taken from Records of The Lord Advocate.

9. Boil suspect material stains in distilled water and then add carbonate of lead and further boil. Collect the precipitate and add dilute nitric acid. Collect the residue, wash with distilled water and decompose by a stream of sulphuretted hydrogen gas for half an hour. Immediately boil and filter the mixture. There will then be in solution free sulphuric acid, the presence of which can be proved by adding nitrate of baryta and a few drops of nitric acid.

10. For detecting free sulphuric acid in stomach contents filter the fluid and then place in a mattrass. Distil with gentle heat and then subject to test 9.

11. Sulphuric acid will turn congo paper blue.

12. Methyl violet will change to blue, then green and with strong acid to yellow

13. Place a drop of barium chloride solution on a drop reaction paper followed by a drop of freshly prepared sodium rhodizonate solution. A reddish brown spot appears which will become decolourised if sulphuric acid is added.

14. Concentrated sulphuric acid will produce charring when added to sugar.

15. To suspect liquids add barium chloride solution. A white precipitate insoluble in hydrochloric acid is formed if suspect liquid is sulphuric acid.

APPENDIX 6

Historical Tests Employed For Detecting Oxalic Acid¹³³

1. Dissolve any suspect crystals in water; ascertain acidity by taste or litmus paper and then precipitate with lime water or solution of chloride or calcium. The precipitate will be insoluble in an excess of the acid, but soluble in nitric acid, if the acid is oxalic.
2. If only solution remaining neutralise with alkali and calcium chloride solution. Any precipitate formed will be soluble in nitric acid, if solution is oxalic acid.
3. If only contents of stomach or vomited matter then boil with distilled water, decolourise with chlorine, filter and add calcium chloride solution. Any precipitate formed will be soluble in nitric acid, if solution is oxalic acid.
4. Oxalic acid will precipitate gold from its solution when boiling.
5. Nitrate of Silver Test. Add silver nitrate and a precipitate of silver oxalate is obtained which if dried and heated on the point of a spatula becomes brown at the end and fulminates with a white fume if oxalic acid is present.
6. Hydrochlorate (Muriate) of Lime Test. Add hydrochlorate of lime to suspect solution and a white precipitate, oxalate of lime, will be formed if oxalic acid present. The precipitate will dissolve on addition of a drop or two of nitric acid.
7. Sulphate of Copper Test. Sulphate of copper when added to oxalic acid will cause a bluish-white precipitate to form.
8. Oxalic acid crystals have the appearance of flattened six sided prisms, which are transparent and free of odour.

¹³³ Note

Tests 1, 2, 5, 6, 7, 12 – Christison, Robert, *A Treatise on Poisons In relation to Medical Jurisprudence, Physiology and the Practice of Physic*, Adam Black & Langam, Rees, Orme, Brown & Green, London, 1829, pp 141-142.

Tests 3, 4, 8 – Thomson, A.T., “Lectures on Medical Jurisprudence” *Lancet*, 1836-1837, p389.

Test 10 – Cowan, H, “Report of the Trial for Mary Struth for Poisoning with Oxalic Acid, with Remarks”, *EMJ*, vol 18, 1862-1863, p94.

Tests 9, 11, 13, 14, 15, 16 are taken from Records of The Lord Advocate.

- 9.** When in pure state oxalic acid will be precipitated in singularly, beautiful, stellated crystals by caustic ammonia. Crystals are radiated star shapes.
- 10.** Oxalic acid crystals when heated on a platinum capsule with sulphuric acid will give off an inflammable gas without blackening and leave a white residue.
- 11.** Oxalic acid crystals when heated in a platinum crucible will leave a greyish-white alkaline ash, which will effervesce with hydrochloric acid.
- 12.** Turns blue litmus paper red.
- 13.** Addition of a solution of calcium salt to suspect solution will produce a white precipitate insoluble in ammonia and acetic acid, but soluble in hydrochloric acid if oxalic acid present.
- 14.** Permanganate Test. Oxalic acid dissolved in hot dilute sulphuric acid, but not boiling, rapidly decolourises a solution of purple potassium permanganate.
- 15.** Oxalic acid discharges the colour of some dyes and slowly reddens others. It does not destroy material as readily as mineral acids.
- 16.** Oxalates evolve carbon monoxide and carbon dioxide when heated with concentrated sulphuric acid.

APPENDIX 7

Historical Tests Employed for Detecting Nitre¹³⁴

1. Dissolve suspect crystals in distilled water and if nitre solution will give a vivid red reaction with brucia.
2. Acid taste on clothes stains.
3. Congo paper turns blue with nitre solutions.
4. Methyl violet changes to blue, then green and with strong acid solution to yellow.
5. Add few drops of diphenlyamine sulphate solution to suspect solution and with care pour mixture upon concentrated sulphuric acid, free from nitric acid. If nitric acid is present a blue zone appears at junction of the two fluids.
6. Mix suspect liquid with an equal volume of concentrated sulphuric acid and cool. Add as an upper layer a strong solution of ferrous sulphate. In presence of nitric acid a brown ring forms at interface.
7. To the suspect sample in sodium hydroxide solution add (0.1 grams) aluminium powder or zinc dust and heat. If ammonia given off (test-mercurous nitrate paper turns black) then nitrate or nitrite present.
8. Heat suspect solution in a test tube with concentrated sulphuric acid and copper filings. If nitrate present there will be reddish fume of nitrogen oxides given off.
9. Mix 1 millilitre of solution to be tested with 3 millilitres of concentrated sulphuric acid and cool. Add a trace of brucine and if nitre solution a red colour, which changes to orange, will appear.
10. Acidify stomach contents. On addition of potassium iodide and starch solution the acid solution becomes deep blue if nitre present.
11. Reddening of litmus paper with nitre solution.
12. Nitre crystals are prismatic and fluted.

¹³⁴ Note

Tests 3, 4, 5, 7, 8, 9 - Glaister, John, *Medical Jurisprudence and Toxicology*, 12th edn, E & S Livingstone, Ltd, London, 1962. Tests 12, 13, 14, 15 – Taylor, Alfred, Swaine, *On Poisons in Relation to Medical Jurisprudence and Medicine*. 2nd edn, John Churchill, New Burlington Street, London, 1859, pp 283 – 284.

Tests 1, 2, 6, 10, 11 are taken from Records of The Lord Advocate.

- 13.** On boiling suspect fluid with some copper cuttings red fumes of nitric acid vapour will be given off leaving a blue solution if nitric acid present.
- 14.** A streak made on white paper with the suspect fluid will not carbonise when heated if nitric acid is present but a visible yellow stain will be left on the paper.
- 15.** Neutralise suspect fluid with potash and then evaporate slowly to form crystals. If crystals appear in the form of lengthened, fluted prisms then nitric acid is present.

APPENDIX 8

Historical Tests Employed For Detecting Carbolic Acid¹³⁵

1. Characteristic smell of carbolic acid in the viscera.
2. Millons' Test. A red colour is produced when a dilute solution of phenol is warmed with Millon's Reagent.
3. A drop of ferric chloride will give an intense blue-violet colour with carbolic acid.
4. A solution of carbolic acid, made alkaline with a quarter of ammonia, develops a transient blue colour on addition of a few drops of bleaching powder (calcium hypochlorite).
5. Add a drop of 10% sodium nitrite solution to 10cc of the suspect solution. Pour the mixture over concentrated sulphuric acid and if carbolic acid is present two layers are formed. One emerald green and the other ruby red.
6. Carbolic acid yields a corpus precipitate of tribromophenol when an excess of bromine water is added.
7. Collect above precipitate, wash and heat gently in a test tube with sodium amalgam and water. Pour mixture into a large watch glass, acidulate and if carbolic acid present there will be a characteristic odour of phenol.
8. Heat suspect solid matter with phthalic anhydride. Cool melt and add caustic soda solution. If carbolic acid present a red colour should be obtained.
9. Carbolic acid will reduce both Fehling's solution and Benedict's solution.
10. Crystals are colourless interlaced needle with rapidly become pink on exposure to air.

¹³⁵ **Note**

Tests 2, 5, 6, 11, 12 – Autenrieth, Wilhelm, *Laboratory Manual for the Detection of Poisons & Powerful Drugs*, 5th edn, translated by Dr William H. Warren, P. Blakison's Son & Co., 1012 Walnut Street, Philadelphia, USA, 1921, p6, p27, p30.

Test 3 – Glaister, John, *Medical Jurisprudence & Toxicology*, 12th edn, E & S Livingstone Ltd, London, 1962, p692.

Test 9 – Polson, C.J., & Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London, 1973, p57.

Tests 1, 4, 7, 8, 9, 10 are taken from Records of The Lord Advocate.

11. Ferric Chloride Test: Very dilute ferric chloride solution, added drop by drop, imparts a blue-violet colour to aqueous carbolic acid solutions. The addition of dilute hydrochloric acid will change this colour to yellow.
12. Hypochlorite Test: Add a few drops of ammonium hydroxide solution to the suspect solution and then 2 or 3 drops of calcium or freshly prepared sodium hypochlorite solution. Gentle warming will produce a blue colour if carbolic acid is present. Very dilute carbolic acid solutions after some time only give a green to blue-green colour.

APPENDIX 9

Sulphuric Acid Post-Mortem Appearances¹³⁶

- 1.** Irritation, inflammation, corrosion of skin.
- 2.** Burning of lips, chin, and front of chest and hands.
- 3.** Corrosion or severe inflammation of larynx and trachea.
- 4.** Inside of mouth shrivelled and brownish.
- 5.** Brownish marks on outside of mouth and linear burns coursing down from angles of mouth.
- 6.** Gums and parts of inside of lips of an almost milky whiteness.
- 7.** Roof of mouth with a glazed appearance and of a greyish-white colour.
- 8.** Uvula ash-coloured.
- 9.** Loss of investing membrane on tongue.
- 10.** Epiglottis membrane ash-coloured and detached in some places.
- 11.** Gullet dry and divested.
- 12.** Erosions or ulceration of the stomach in branched or winding furrows.
- 13.** Cardiac orifice of stomach hard and contracted.
- 14.** Stomach converted into soft, baggy, black mass which readily disintegrates when touched.
- 15.** Arch of stomach corroded and destroyed.
- 16.** Lower part of duodenum red on inner surface.
- 17.** Intestines distended and highly inflamed on the peripheral surface.

136 Note

Aparances 1, 2, 3, 15 – Polson, C.J., & Tattersall, R.N. *Clinical Toxicology*, 2nd edn, Pitman, London, 1973, pp40-41.

Aparances 4, 5 – Ryan, M, “Homicide by Poisoning”, *London Medical & Surgical Journal*, vol 6, 1831, p405.

Aparances 6, 7, 8, 9, 10, 11, 12, 16 – Christison, Robert, “Cases and Observations in Medical Jurisprudence”, *EMJ*, vol 35, 1831, pp302-303.

Aparances 13, 14 – Thomson, A.T, “Lectures on Medical Jurisprudence”, *Lancet*, vol 2, 1836-1837, p360.

Aparance 17 – Sinclair, Martin, “Cases of Poisoning by Sulphuric Acid”, *EMJ*, vol 36, 1831, p101.

Aparances 18, 19, 20, 21, 22 are taken from Records of The Lord Advocate.

- 18.** Bowels inflamed.
- 19.** Rima glottides contracted.
- 20.** Blackening of tissue burns.
- 21.** Skin becomes parchmented after death.
- 22.** Burns simulating abrasions.

APPENDIX 10

Oxalic Acid Post-Mortem Appearances¹³⁷

- 1.** Upper extremities, chest and back much discoloured.
- 2.** Abdomen turbid.
- 3.** Mucous membrane of pharynx and oesopahagus appear as if been scalded and easily separated from muscular coat.
- 4.** Villous coat of stomach pulpy and in many places black.
- 5.** Patches of extravasated blood in large intestine.
- 6.** Peritoneal coat of stomach and intestines much inflamed.
- 7.** Mucous membrane of larynx, trachea and lungs much inflamed.
- 8.** Stomach distended.
- 9.** Mucous membrane of tongue hardened and blackened.
- 10.** Inflammation of intestines.
- 11.** Inflammation of colon.
- 12.** Extensive inflammation of all mucous membranes.
- 13.** Lips of a blackish colour and exhibiting corroded appearance.
- 14.** Whitening or yellow-white discoloration of lips, lining of mouth and upper surface of tongue.
- 15.** Stomach perforated.

¹³⁷ **Note**

Appearance 3 – Christison, Robert, *A Treatise on Poisons in Relation to Medical Jurisprudence, Physiology & the Practice of Physic*, Adam Black & Longman, Rees, Orme, Brown & Green, London, 1829.

Appearances 4, 5, 13, 15, 16, 18, 19 – Littlejohn, Henry, Duncan, “case of Criminal Poisoning with Oxalic Acid in which Perforation of the Stomach Took Place”, *EMJ*, vol 7, 1868, p14.

Appearances 8, 9 – Christison, Robert, “Processes for Detecting Poisons”, *Lancet*, vol 1, 1830-1831, p418.

Appearance 17 – Polson, C.J., & Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London, 1073, p71.

Appearance 10 – Cowan, H, “Report of the Trial of Mary Struth for Poisoning with Oxalic Acid, with Remarks”, *EMJ*, vol 18, 1862-1863, p98.

Appearances 1, 2, 6, 7, 11, 12, 14, 17, 20, 21, 22 are taken from Records of The Lord Advocate.

- 16.** Corrosion of mucous membranes of upper digestive tract.
- 17.** Mucosa of stomach blackened.
- 18.** Stomach in a state of decomposition, soft and friable and tearing on attempts to ligature it.
- 19.** Gastric content dark brown colour with fresh and altered blood.
- 20.** Upper portions of intestines inflamed.
- 21.** Octahedral crystals found in stomach.
- 22.** Crystals deposited in the urinary tubules.

APPENDIX 11

Nitre Post-Mortem Appearances¹³⁸

1. Congestion of the mucous membrane at the lower end of the gullet where it joins the stomach.
2. Right cavities of heart distended with dark fluid blood.
3. Internal surface of stomach bright cherry red colour.
4. Small intestines much corrugated and congested on their external surface.
5. Lining membrane of stomach of a brownish-red colour, generally inflamed, and in parts detached from coat beneath.
6. Bloody mucus in stomach
7. Numerous erosions in stomach.
8. Inner surface of stomach coated with large quantity of glary and sanquionolent matter.
9. Lesions in digestive organs.
10. Skin of lips excoriated; that of tongue yellow.
11. Lungs blackish or violet colour.
12. Small intestines corrugated and congested on external surface.

¹³⁸ **Note**

Aparances 1, 2, 3, 4, 12 – Littlejohn, Henry, Duncan, “The Practice of Medical Jurisprudence with Hints as to the Conduct of Medical Practitioners in Cases of Suspected Poisoning”, *EMJ*, vol XXXI, 1885, p103.

Aparances 5, 6, – Taylor, Alfred, Swaine, *On Poisons in Relation to Medical Jurisprudence and Medicine*, 2nd edn, John Churchill, New Burlington Street, London, 1859, p338.

Aparances 7, 8, 9, 10, 11 are taken from Records of The Lord Advocate.

APPENDIX 12

Carbolic Acid Post-Mortem Appearances¹³⁹

- 1.** Stains at the angles of the mouth and on the chin.
- 2.** Mucous membrane of mouth softened and either white or ash grey in colour.
- 3.** Peritoneal surface of stomach infected.
- 4.** Mucous coat of stomach usually corrugated, toughened and of a greyish-white colour.
- 5.** Staining and hardening of liver and spleen.
- 6.** Parenchymal degenerative changes in kidneys.
- 7.** Severe corrosion in stomach.
- 8.** Stomach hardened with a leathery feel.
- 9.** Partial separation of necrotic mucosa of stomach.
- 10.** Coagulation necrosis of mucosa and severe congestion of submucosa.
- 11.** Laryngeal and pulmonary oedema.
- 12.** Corrosion of skin especially in tracks from angles of mouth.

¹³⁹ **Note**

Aparances 2, 3, 4, 5, 6, 7, 9, 11 are taken from Records of The Lord Advocate.

Aparances 1, 8, 10, 12 - Polson, C.J., & Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London, 1973, p59.

Chapter 4

Strike Up A Light Or Lucifer's Element

4.1 Introduction

Phosphorus is a non-metallic element, the thirteenth element in the periodic table. It does not occur naturally, only in the oxidised form of phosphate, Phosphorus may occur in crystalline, mixed crystalline and amorphous form. The former, which is yellow phosphorus, is usually found commercially as yellow, translucent, waxy-looking sticks, and usually kept submerged in water to prevent oxidation. This yellow phosphorus, sometimes called white phosphorus, is poisonous. The mixed form is red phosphorus and is not poisonous.¹

The word phosphorus is derived from the ancient Greek word *phos*, meaning “light” and *phorous*, meaning, “bringing.” It is alleged to have been discovered in 1669 by the Hamburg alchemist, Henning Brandt, in his search for a compound that would turn base metals like lead into gold.² Frustrated by his inability to make gold with phosphorus after six years, Brandt finally revealed its existence to other chemists who began their own experiments with the new element.³

By the early 1800s many influential medical men had voiced their support for the curative powers of phosphorus, and yellow phosphorus began to be used in medicine.⁴ It was suggested that in very small doses it was useful in the treatment of bone

¹ Glasiter, John, *Medical Jurisprudence and Toxicology*, 12th edn, E & S Livingstone Ltd, London, 1962, p538.

² “Like many before him Brandt had been investigating the golden stream, urine, and he was heating the residues from this, which he had boiled down to a dry solid. He stoked his small furnace with more charcoal and pumped the bellows until his retort glowed red-hot. Suddenly something strange began to happen. Glowing fumes filled the vessel and from the end of the retort dripped a shining liquid that burst into flames. When he caught the liquid in a glass vessel and stoppered it he saw that it solidified but continued to gleam with an eerie pale-green light and waves of flame seemed to lick its surface. It continued to shine undiminished hour after hour.” – Emsley, J., *The Shocking History of Phosphorus*, Macmillan Publishers, London, p1.

³ Ibid, p5.

⁴ Ibid, p52

disorders, neuralgia, and even tuberculosis.⁵ Indeed, phosphorus did not disappear from the *British Pharmacopoeia*, the official listing of all prescribed medicaments, until 1932, and was still available as an over the counter remedy in the UK in the 1950s.⁶

When swallowed in excessive doses however, phosphorus causes pains in the stomach, which are usually followed by vomiting.⁷ A garlic taste may develop in the mouth and there is an acrid, burning sensation in the throat and oesophagus, accompanied by great thirst. Signs of shock develop, the pulse becomes small, irregular, feeble, and, at times, imperceptible and the skin becomes cold and clammy. There is usually an intermission in the severity of symptoms which suggests that recovery has taken place but, in fatal cases eventually there are convulsions or coma and then death. There are instances of recovery on record however for poisoning with phosphorus.⁸

⁵ Ibid, pp51 – 58.

⁶ Elemental phosphorus could be purchased in many forms: dispersed in olive oil, turpentine or mixed with cod liver oil or mixed with beeswax. There was also phosphorated water, soda water or vinegar. It was sold as a popular remedy for toothache, neuralgia and was also a popular aphrodisiac and tonic. See Emsley, J., *The Shocking History of Phosphorus: A Biography of the Devil's Element*, Macmillan Publishers, London, 2000, pp47 – 63. Externally yellow phosphorus was used in “cancer cures”, generally made up into the form of a paste. In small doses, for a short period, it was meant to stimulate stomach functions and so improve digestion. Taken over longer periods, it was used for chronic skin diseases, in various nervous diseases, in pernicious anaemia, in asthma and chronic malaria – Comrie, John, D., *Black's Medical Dictionary*, 8th edn, A & C Lack Ltd, London, 1926.

⁷ Glaister, J., *Medical Jurisprudence and Toxicology* 12th edn, E & S Livingstone Ltd, 1962, p539.

⁸ Emsley, J., *The Shocking History of Phosphorus: A Biography of the Devil's Element*, Macmillan Publishers, London, 2000, p190 –“remarkable recoveries are on record such as the man who spooned half a tin of Rodine rat poison into a tumbler of whisky and drank it in a suicide attempt. This gave him a dose of around 300mg enough to kill most people. Within two hours he was admitted to Guy’s Hospital in London suffering from extreme stomach pains and vomiting; his vomit smelled of phosphorus. He was given emetics for several hours until his vomit no longer smelled of the poison. Three days later his liver started to enlarge and jaundice set in, but on the sixth day his condition began to right itself and eventually he recovered.”

4.2 The Uses and Abuses of Phosphorus

Since 1669 the development of the destructive potency of phosphorus has been profound.⁹ It has often been called the Devil's Element. It is, therefore, of little surprise that in 1869 the idea was put forward that phosphorus had taken the place of arsenic as the most popular toxic agent of the time and that it had become the poison of choice for many.¹⁰ This alleged substitution of phosphorus for arsenic was, no doubt brought about by the Arsenic Act of 1851 and the subsequent introduction of phosphorus pastes in the nineteenth century which were designed for the destruction of rats, mice and other noxious vermin. Ironically, this was widely welcomed as a "safety measure". Naturally however, just like arsenic, these new rat poisons could be just as effective when used for criminal purposes.¹¹

The accessibility of this poison to the public was made even greater by the use of phosphorus in Lucifer matches from the 1830s until 1910 when legislation was finally passed prohibiting the use of white phosphorus for this purpose.¹² These matches

⁹ Phosphorus was used extensively throughout the Second World War in bombs. In particular, for operation Gomorrah, the destruction of Hamburg with bombs containing phosphorus, where at least 37,000 people were killed over five days and nights. It was also used in the manufacture of potential war gases and by the end of the Second World War it was discovered that the Nazis had manufactured enough Tabun (phosphorus based gas) to exterminate all human life on earth. After the Second World War organophosphorus nerve gases were made, such as Sarin, which was used by the Iraqi Government in 1998 to kill Kurdish villagers and by the AWM Shinrikyo Sect on the Tokyo underground on 19th April 1995. It has also been reported on numerous occasions that Israel's military have fired artillery shells with the incendiary agent white phosphorus into Gaza in contravention of the Geneva Treaty – See Times Online, 5th January 2008, Franklin, S., *Israel Rains Fire on Gaza with Phosphorous Shells*; Pakistani Times, 2nd February 2009, Azam, O., *Pakistan Islamic Medical Association Witnesses Phosphorus Bombing on Gaza*.

¹⁰ Jenkins, T.E., *Half Yearly Abstract of the Medical Sciences*, Vol L, 1869, p89, On Poisoning by Phosphorus.

¹¹ The sale of vermin poisons, containing yellow phosphorus, ended in the UK in 1963 when the Animals (Cruel Poisons) Regulations came into force.

¹² History records three claims to the invention of phosphorus matches in the 1830s by Charles Saucra of France, Jacob Kramer of Germany and Stephen Rommer of Austria. A typical Lucifer match head contained 20% white phosphorus, 15% sulphur, 30% potassium chlorate, 10% chalk and 25% glue.

were poisonous if sucked, often putting very young children at risk.¹³ Further, these matches were often the common agents of adult suicides.¹⁴ Although one match head contained too little phosphorus to be effective as a poison, packs of a dozen boxes could be purchased in the 1880s for only one penny.¹⁵ White phosphorus was, therefore, widely and cheaply available to all.¹⁶

The Victorians believed that taking phosphorus would improve mental ability and that it acted as an aphrodisiac. The fact that phosphorus could be chemically extracted from urine and also glowed with its own source of light only added to its attraction.¹⁷

¹³ See Sinclair, Alex, J., *EMJ*, vol 32, 1887, pp 50-51, Case of Phosphorus Poisoning. This article reports the case of a 22 month child who sucked between a dozen and a half to two dozen heads of yellow phosphorus matches of the variety called “Ruby” and manufactured by Bryant and May. She died within 24 hours.

¹⁴ Ogston, Francis, *EMJ*, vol 7, 1861-1862, p581, Poisoning by Lucifer Matches.

¹⁵ The largest manufacturer of British matches was Bryant and May. They produced Lucifer matches under the brand names of “Pearl”, “Tiger” and “Ruby” and by 1884 employed some 3,000 people. Working conditions for matchmakers were, however, appalling and many suffered what is known as phossy jaw, due to yellow phosphorus poisoning. With phossy jaw the teeth and gums would become eroded to such an extent that often the jaw bone was completely eaten through. See Act 1, Scene 1 of the musical “The Matchgirls” by Bill Owen and Tony Russel in 1888 -“Top grade selectable; Hardly detectable; Phosphorus, phosphorus. Taste is more subtler and spreads just like butter – grand phosphorus, phosphorus. Our special beauty cream we look a proper dream – for we are minus a jaw. Guv’nors don’t charge a fee; Give it away for free, phosphorus, phosphorus, phosphorus.” Note also one of the saddest sights in the Odontological Museum of the Royal College of Surgeons of England is the display case that preserves the jawbones of some of the match girls.

¹⁶ The matter could be scraped off from the points of the Lucifer matches and dissolved in water or brandy.

¹⁷ The ability of phosphorus to glow in the dark, known as luminescence, is due to the slow chemical reaction between phosphorus and the oxygen of the air which takes place on the surface of phosphorus, forming two species which have only a fleeting existence: a molecule of formula HPO and an oxide of formula P₂O₂. Both emit visible light. Very little of these unstable species need be formed to produce the luminescence, which is why a piece of phosphorus in a closed vessel continues to glow for hours and days, until the last trace of oxygen has been used up – See Rawcliffe, C.T., and Rawson, D.H., *Principles of Inorganic and Theoretical Chemistry*, 2nd edn, Heinman Educational Publishers, 1969, pp261-263.

This glow was taken as strong evidence that phosphorus was the *flammula vitae*.¹⁸ The availability of medicines and matches containing white phosphorus inevitably produced fatalities, and people often accidentally overdosed.¹⁹ The ease of purchasing phosphorus was further aided by lack of legislation. Not until the Pharmacy and Poisons Act of 1933 was the sale of phosphorus compounds controlled by the legislature.²⁰ Until then, however, the public could lawfully acquire a substantial amount of phosphorus for only a few pence.

Phosphorus had a distinct advantage as a murder weapon. The devastation produced in the body caused by exposure to phosphorus was consistent with liver disease or gastritis.²¹ It was, therefore, possible for a person with criminal intent to stimulate more or less perfectly an internal illness, inflammatory or otherwise, by administration of phosphorus in food or liquid medium.²² Hence two unfortunate results could be realised: a very execrable crime would remain unpunished, and doctors misled as to what illness they were treating.

Thus far, the medico-legal implications of phosphorous would seem to be similar to those in the cases investigated in arsenic poisoning. However, it was often impossible in the past to detect phosphorus even in the remains of someone who was known to

¹⁸ The Flame of Life. The famous painting *The Alchymist In Search of the Philosophers Stone, Discovers Phosphorus* by Joseph Wright of Derby (1734-1797), which is in the Bridgeman Art Library, captures the wonder of the discovery of the impressive glow from phosphorus.

¹⁹ Ogston, F., *EMJ*, vol 7, 1861-1862, p581, Poisoning by Lucifer Matches; Sinclair, Alex, *EMJ*, vol 32, 1887, pp800-801, Case of Phosphorus Poisoning.

²⁰ See Pharmacy and Poisons Act 1933, First Schedule.

²¹ Poulet, M., *Half Yearly Abstract of the Medical Sciences*, vol LV1, July – December 1872, p110, On the Diagnosis of Yellow Phosphorus Poisoning by Means of a Sign Furnished by the Urine of the Patient.

²² Phosphorus does, however, have a very distinctive garlicky taste and smell, which is difficult to disguise.

have died from phosphorus poisoning.²³ Thus, suspicion of phosphorus poisoning and the ability to supply proof to the satisfaction of a court of law were two very different propositions. Given these considerations, the anecdotal accounts mentioned earlier of the popularity of phosphorus as a poison following the Arsenic Act of 1851, seem likely to have accurately reflected social fact.

Phosphorus, however, was not, in many of its available manifestations, an ideal poison. Although freely available in medicines, matches and rat poisons, the medicines contained too little phosphorus to be effective and matches were contaminated with other agents that a potential victim might recognise.²⁴ Thus, as with arsenic, it seems likely that it was often rat poison that poisoners chose. Tins of rat poison containing phosphorus were easily obtained and not expensive.²⁵

The difficulty in using phosphorus to kill a person, as opposed to a rat, lay in disguising its strong garlicky smell and flavour from the intended victim. Records for only two trials for poisoning were discovered in the course of this research only 3.5% of all my cases and, though one of these cases occurred after 1911, the circumstances of the case are much the same as they would have been in the previous fifty years. This figure is comparable with the English figure of only three cases during the period 1750-1914 and certainly between 1739 and 1878 no cases of poisoning with

²³ This is because phosphorus only becomes poisonous in becoming phosphorus or phosphoric acid by combining with oxygen. Thus, in many cases of phosphorus poisoning when vomited matters etc..., were not kept and placed in some fluid that preserved the phosphorus unchanged, chemical detection would fail. Also inflamed portions of the stomach and intestines might not have given rise to phosphorescence (a process whereby light is first absorbed by a body and then re-emitted from it some time later) and the reactions significant of the presence of phosphoric acid and phosphates would not justify an expert in affirming the presence of phosphorus.

²⁴ See note 12. In addition, lead dioxide or nitrate was sometimes added to help ignite such matches.

²⁵ The phosphorus rat poison was usually a paste containing a compound of yellow phosphorus, sugar and bran. The most popular brand was Rodine, which consisted of a paste of bran and molasses with 2% phosphorus. One tin of Rodine contained 650mg of phosphorus. It is generally accepted that the minimum lethal dose of yellow phosphorus is 60mg and that as little as 15mg can cause ill-effects -Goodman, L.S., and Gilman, A., *The Pharmacological Basis of Therapeutics*, 2nd edn, Macmillan, New York, 1955, pp821-822. It has been said that even 7mg proved fatal – Witthaus, R.A., *Manual of Toxicology*, 2nd edn, Bailliere, Tindall & Cox, London, 1911, p635. One teaspoonful of Rodine would, therefore, have provided a fatal dose.

phosphorous were recorded at the Old Bailey.²⁶ Given the widespread availability of phosphorus it seems likely that there would have been many more attempts, both successful and unsuccessful, to dispose of others by means of phosphorus poisoning.

4.3 Phosphorus Poisoning Cases

The first case concerns William Dallas Coull of Montrose, who, having fallen out with his wife, Helen, had left their marital home to stay with his parents during the Christmas and New Year of 1896/7.²⁷ On 2nd January 1897 a neighbour handed Helen a paper bag. The bag, which bore the name of a local baker, contained fancy bread and cakes and had been delivered to the neighbour by a young boy whilst Helen was out. On opening the bag Helen noticed a strange smell and after putting a small piece of cake in her mouth felt it had an offensive taste.²⁸ She immediately examined the contents of the bag and saw that the sandwich cake had been smeared with what appeared to be vermin paste. The police were called and told of the circumstances surrounding the delivery of the cakes. Further enquiries produced evidence that Coull had bought the cakes at the bakers that day, and then asked ten year old James Henderson to take them to his wife.²⁹ Coull was, therefore, arrested and charged with attempting to murder his wife.

Coull's trial was held in Dundee on March 24th 1897, where the Crown's case was bolstered by the forensic evidence given by Dr Harvey Littlejohn. The sandwich cake had been found to contain 0.35 grains of phosphorus.³⁰ In addition, a mass of greenish colour on the sandwich cake was proved at trial to be identical to the contents of a bottle of 'Steiner's Vermin Destroying Paste'.³¹ This bottle had been removed from

²⁶ See Watson, K., *Poisoned Lives: English Poisoners and their Victims*, Hambledon and London, London, 2004, p33; Forbes, *Surgeons at the Old Bailey*, Yale University Press, London, 1985, Table 8.

²⁷ This falling out was allegedly due to his drinking and refusal to financially support his wife and baby daughter – Record of the Lord Advocate AD14/97/77.

²⁸ Helen immediately spat the piece of cake out.

²⁹ Coull had told James that he had got the bag of cakes from a lady and requested James to tell Helen that a lady had sent the cakes.

³⁰ Appendix 14, tests 4, 5,6,7,8.

³¹ Appendix 14, tests 4, 5,6,7,8.

the home of Coull's parents.³² At trial Coull pleaded not guilty. All the main circumstances were present, however, on which reliance can be placed for establishing guilt.³³ Despite this, the jury returned a unanimous verdict of 'Not Proven'.

It would seem that an unsatisfactory verdict was returned in this case. The verdict of the trial depended almost completely on the chemical evidence. Given that there was no dead body, no evidence to indicate that Coull himself had purchased rat poison, and very little drama, it would appear that this Victorian jury were unwilling to convict on mere medical testimony alone. Indeed, in eighteen Scottish poisoning trials from 1859 onwards until the end of the Victorian era in 1901 only seven of the accused were found guilty (39%).³⁴ In the period preceding that (1800 -1858) in thirty-nine trials, twenty-three of the accused had been found guilty (59%).³⁵

By the mid nineteenth century forensic science was no longer in its infancy and it would appear by then that any jury in a poisoning trial expected reliable tests to have been used to provide definite evidence of poisoning. Further, there were legal difficulties where the victim of an alleged poisoning survived, as in such cases there were no post mortem appearances to present to a jury and of course no samples of poison to produce from the body of a deceased.

Before the start of the nineteenth century a person could theoretically be sentenced to death for the modest offence of theft of a small amount of goods. By the mid nineteenth century, however, attitudes towards crime had changed, and following the introduction of the Criminal Law Consolidation Act 1861, where the number of capital crimes was reduced from approximately eight to four, there appears to have been a certain unwillingness to find an accused guilty in a capital crime unless there

³² Coull's mother had bought the bottle of vermin poison on the 31st December 1896 for 3d. She stated that she had been much annoyed with rats in the house. Only half of the contents of the bottle remained – Record of the Lord Advocate AD14/97/77.

³³ Proof of possession of poison, opportunity for secret administration and motive. Motive was present due to Coull's great anger with his wife for not letting him move back in with her.

³⁴ 7/18 x100 = 39%.

³⁵ 23/39 x100 = 59%.

was absolute certainty.³⁶

The second case concerns Unisbella Fraser, a domestic servant in the home of Mary Aitken Hunter in Grangemouth.³⁷ Between 20th September 1915 and 30th December 1915 Mary Aitken constantly accused her employee of stealing from her and found various articles belonging to her in Fraser's room on the 30th December 1915.³⁸ Fraser admitted the theft and Mary Aitken advised her that she could remain in her employment for a further month, but would then have to leave. Despite this, on Thursday 20th January 1916, Mary Aitken found various household articles hidden under her employee's mattress.³⁹

Next morning Unisbella Fraser prepared tea for her mistress who, on beginning to drink it, noticed an odour of phosphorus.⁴⁰ Whilst Mary Aitken was sipping the tea Fraser entered the room in an excited and angry state. She demanded to be given the teapot and told her mistress that she had no right to search her room, stating: "(Y)ou have no right to go into my room, I am as good as you and better."⁴¹ A violent struggle followed in which Unisbella Fraser held her mistress by the throat and compressed it saying, "(Y)ou will not live to tell the tale."⁴² The arrival of the

³⁶ In 1800 there were 16 capital crimes; however in the period 1832-1847 Sir Robert Peel's government introduced various Bills to reduce the number of capital crimes. Shoplifting, sheep, cattle and horse stealing were removed from the list in 1832, followed by letter stealing, returning from transportation (1835), forgery and coining (1836), wilful fire raising, burglary and theft from a dwelling house (1837) and rape (1841). Attempted murder was reduced from being a capital crime in 1861. The list of capital crimes was finally reduced to: murder, high treason, piracy and wilful fire raising in a royal dockyard.

³⁷ Record of the Lord Advocate AD15/16/15.

³⁸ Unisbella Fraser was alleged to have stolen cake and pastry whilst in her room was discovered a night-gown, a pair of combinations and three chemises belonging to Mary Aitken and her daughter. Record of the Lord Advocate AD15/16/15.

³⁹ Found under the mattress were six pairs of stockings, six handkerchiefs, a pocket book, six photographs, a workbag, scissors, an eyeglass, a birthday book and a necklace. Record of the Lord Advocate AD15/16/15.

⁴⁰ At first Mary Aitken thought that the smell was coming from the local explosives works, which were only a short distance from the house.

⁴¹ Record of the Lord Advocate AD15/16/15.

⁴² Ibid.

milkman, however, at the back door brought an interruption, and soon after Unisbella Fraser was arrested and charged with attempting to poison her mistress.

Mary Aitken appeared to suffer no adverse effects from drinking the tea. Chemical analysis, however, of the contents of the teapot and teacup by local pharmacist Robert Sinclair revealed the presence of phosphorus in both.⁴³ At the trial of Unisbella Fraser the testimony of Robert Sinclair was backed up by that of Dr Harvey Littlejohn and pharmaceutical chemist, J. Rutherford Hill. This was following chemical analysis of the teapot and teacup contents.⁴⁴ Conclusive evidence, therefore, existed that phosphorus had been placed in the teapot. Although it could not be proven in any way that Unisbella Fraser had purchased poison containing phosphorus, two tins of rat poison had been kept in the house.⁴⁵ Following chemical analysis both tins were shown to contain phosphorus.⁴⁶ In addition, it was shown that the tin of “Hailey’s Rodine Rat Poison” appeared to have been recently opened and some of the contents removed.⁴⁷

In this case the accused had access to poison of the particular kind found in the teapot and teacup. Further, opportunity existed for its secret administration, and the motive was Unisbella Fraser’s fear or anger at the dismissal from her job. Supplementary to this was the testimony of Mary Fairley Waddell, a friend of the accused to whom Fraser had confided that she had put poison in the teapot. “I will tell you something if you will not tell anybody else. I put poison in the teapot.”⁴⁸ Despite pleading not guilty to the charge of attempted murder, the panel was found guilty at Edinburgh High Court on April 11th 1916. She was sentenced to three years imprisonment.

⁴³ Appendix 14, tests 4, 6, 8.

⁴⁴ Appendix 14, tests 4,6,8,9.

⁴⁵ There was a tin of “Harrison’s Reliable Rat and Mice Poison” and a tin of “Hailey’s Rodine Rat Poison”.

⁴⁶ Appendix 14, tests 4,6,7,8.

⁴⁷ The contents of the tin of “Harrison’s Reliable Rat and Mice Poison” was shrunken and dried up pointing to the probability that the box had been lying unopened and unused for several years. It was apparent on opening that this tin had not been interfered with recently. On the other hand a large quantity of paste appeared to have been recently removed from the tin of “Hailey’s Rodine Rat Poison.”

⁴⁸ Record of the Lord Advocate AD15/16/15.

In the nineteenth century many fatal encounters between mistresses and maidservants were recorded.⁴⁹ Given that there had been many publicised cases of murder by servants in the press it is perhaps not surprising that Unisbella Fraser was found guilty, unlike William Dallas Coull⁵⁰. Further her confession marks out an important difference between the two cases highlighted in this section.

4.4 Conclusions

Phosphorus was cheap, easy to obtain and above all difficult to detect. Doctors would fail to recognise symptoms, and it was often even impossible to detect any phosphorus at all in the remains of someone known to have died from phosphorus poisoning. As little as one grain can prove fatal. Also given that the first Births, Deaths and Marriages (Scotland) Act was not introduced until 1854; it is possible that many suspicious deaths went unrecorded.⁵¹ Further, many trial transcripts have been lost to history. All these factors contribute to suspicion of the argument that homicidal poisoning was a rare occurrence in the past. It seems unlikely that a poisoner would have ignored such a convenient agent as the Devil's Element. Though cases are rare, the findings of Chapter Three on corrosive acids should also alert us to the fact that the murder of infants could easily have been carried out using undetectable

⁴⁹ See Altick, R.D., *Victorian Studies in Scarlet*, LM Dent & Sons Ltd, 1972, p220. Note also that even lower income householders could afford maidservants up until 1939 as labour was both cheap and plentiful, and that during the nineteenth century that the largest occupation of women was in domestic service. Whilst in most written accounts of domestic life, the large households of the well-to-do feature prominently, because it was there that service was seen at its most spectacular. But at no time during the eighteenth and nineteenth centuries did the bulk of domestic servants work in such establishments. For example it was recommended in 1825 for a household consisting of a gentleman and lady with children and annual income in region of £500 to £600 (around £25,000 in today's money) that there be a cook, a house maid and a nursery maid. See Adams, S., *The Complete Servant*, Southover Press, 1989, p16 , reprinted from Adams S. & Adams, S., *The Complete Servant*, London, 1825.

⁵⁰ Attempted murder of her mistress Christain Ritchie in 1816 by Catherine Clerk; murder of the Duchess of Sutherland in Edinburgh in 1849 by her maidservant Maria Manning; attempted murder of her master Robert Kincaid in 1861 in Stirling by Agnes Kirkwood; attempted murder of her master Thomas Hay by Ellen McLeod or Beaton in 1896 in Elgin.

⁵¹ See Births, Deaths and Marriages (Scotland) Acts, 1854-1860. Following this legislation a doctor's certificate was required to register a death.

phosphorus, and, given the myriad causes of infant mortality, investigation of such cases would be unlikely.

The conclusions of Chapter Three as to the relative weighting given to chemical and circumstantial evidence by Victorian juries, is corroborated by the above phosphorous cases. Though chemical analysis of the means of poisoning (cakes, teapots) proved the presence of phosphorous, circumstantial evidence was relied upon greatly in reaching verdicts. Indeed, the chemical testing was regarded simply as another piece of circumstantial evidence. In the Coull case, though all evidence suggested attempted murder, none of the circumstances of the case, including the analysis of the cakes, was deemed to prove that Coull had tried to murder his wife.

These cases, wherein no deaths were caused, are a difficult sample from which to draw conclusions. Nevertheless it is of little surprise, given the impossibility of detection of phosphorous post-mortem, that these cases are only evidence. This fact, however, does not seem to have been impressed upon the juries in these cases. One might expect an especial desire to punish attempted murder by phosphorous given that murder by phosphorous could not be discovered after death. However, lack of communication between medical and juridical professionals seems to have precluded such a stance. Instead juries simply cast medical analysis as another circumstance of the case and adopted lenient attitudes.

APPENDIX 13
Phosphorus Poisoning Cases 1897 – 1916

Date	Place	Accused	Victim(s)	Medium of Administration	Excuse for Obtaining Phosphorus	What Happened to Victim(s)	Trial Verdict
1897	Montrose	William Dallas Coull	Wife - Helen Coull	In Cakes	For Killing Rats	Lived	Not Proven
1916	Grangemouth	Unisbella Fraser	Mary Aitken	In Tea	None Given	Lived	Guilty-sentenced to 5 Years

APPENDIX 14

Historical Tests Employed For Detecting Phosphorus⁵²

1. Phosphoric odour of garlic.
2. Sherer's Test. Heat suspected material in a flask with dilute acid over, which has been placed filter paper wetted with silver nitrate solution. If the rising steam causes the filter paper to turn black, phosphorus is present.
3. Extract suspected materials from vomited matter or stomach contents using carbon disulphide on filter paper and allow solvent to evaporate. If phosphorus is present the paper will glow in the dark.
4. The taste of phosphorus is reminiscent of garlic or celery.
5. When exposed to air and light phosphorus evolves a white vapour.
6. In the dark phosphorus shines with a faint bluish light (phosphorescence).
7. Phosphorus burns with a yellow flame producing dense white fumes.
8. Mitscherlich's Test. Mix suspected material with water acidified with sulphuric acid and transfer to a glass retort fitted with a long condenser. The tube from the retort is fitted with an adapter, which ends in a receiver containing a solution of silver nitrate. Distillation is made in the dark, luminosity in the tube being evidence of the presence of phosphorus.
9. Ammonium Molybdate-Benzidine Test. Place a drop of suspect solution on filter paper and add a drop of molybdate reagent. Place into 35ml of concentrated nitric acid and add a drop of benzidine reagent. On holding wetted filter paper with this solution over ammonia vapour a blue stain is formed if phosphorus is present.
10. Phosphorus is insoluble in water.

⁵² Note

Tests 1, 5, 6, 7, 10 – Taylor, Alfred, Swaine, *On Poison in Relation to Medical Jurisprudence and Medicine*, 2nd edn, John Churchill, New Burlington Street, 1859, pp349-350.

Tests 2, 8, 9 – Glaister, John, *Medical Jurisprudence & Toxicology*, 12th edn, E & S Livingstone Ltd, London, 1962, pp692-693.

Tests 3, 4 – Couseran, M, “On the Detection of Phosphorus”, *EMJ*, vol 6, 1860-1861, pp387-391.

APPENDIX 15

Post-Mortem Appearances⁵³

- 1.** Irritation of the gullet and intestines.
- 2.** Irritation of the stomach.
- 3.** Liver enlarged and yellow in colour.
- 4.** Stomach distended with gas, which smells of garlic.
- 5.** Corrosion and perforation of the gastric-intestinal tract.
- 6.** Ulceration of the stomach.
- 7.** Fatty degeneration in heart and kidneys.
- 8.** Gastric and intestinal contents smell of sulphur or garlic and are luminous.
- 9.** Skin generally yellow in colour.
- 10.** Phosphorescence of inflamed portions of stomach and intestines.

53 Note

Aparances 1, 2, 8 – Taylor, Alfred, Swaine, *On Poisons in Relation to Medical Jurisprudence and Medicine*, 2nd edn, John Churchill, New Burlington Street, London, 1859, pp346-347.

Aparances 3, 4, 5, 6, 7 – Polson, C.J., & Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London, 1973, p295.

Aparance 9 – Christison, Robert, *A Treatise on Poisons in Relation to Medical Jurisprudence, Physiology and the Practice of Physic*, Adam Black and Longman, Rees, Orme, Brown & Green, London, 1829, p135.

Aparance 10 – Couseran, M, “On the Detection of Phosphorus”, *EMJ*, vol 6, 1860-1861, p387.

Chapter 5

Prescriptive Poppy Or Pestilent Poison

5.1 Introduction

Opium is a plant product - the brown tacky substance which is obtained after drying the milky exudate which oozes when the poppy capsule is incised. Its effects on the human mind have probably been known for about six thousand years, and it had a significant place in Greek, Roman and Arabic medicine.¹ This material which, eaten, made up into a drink, or smoked, provided the drug in effective form over the millennia, and it was only in this traditional plant form that the drug was available at the start of the nineteenth century. The opium poppy – the species cultivated for opium production – is *Papaver Somniferum*, a white poppy growing to a height of about one or two feet. The poppy is, and has been, grown chiefly in Asia Minor, China, Iran and some Balkan countries.

In the nineteenth century, many preparations based on opium, or patent remedies with opium as their active ingredient, were widely available for purchase. Among the best known and most widely used were: *Laudanum*; *Paregoric*; *Batley's Sedative Solution*; *Dover's Powders*; *Godfrey's Cordial*; *Mrs Winslow's Soothing Syrup*; *Atkinson's Infant's Preservative*, and *Street's Infants Quietness*.² Self medication with opium was widespread as it was easily purchased, as well as being prescribed by doctors.³

¹ Berridge, V., *Opium and the People*, Free Association Books Ltd, London, 1999, pXV111.

² Ibid. Laudanum was otherwise known as tincture of opium, made by mixing opium with distilled water and alcohol; paregoric was camphorated tincture of opium (the word paregoric being derived from the Greek word of soothing); Batley's Sedative Solution was opium mixed with calcium hydrate, alcohol, sherry and water; Dover's Powders consisted of opium, saltpetre, tartar, liquorice and ipecacuanha. Godfrey's Cordial was a "children's opiate" based on laudanum, as were Mrs Winslow's Soothing Syrup, Atkinson's Infants' Preservative, and Street's Infants Quietness.

³ The populace could purchase opium gout remedies, corn plasters, children's draughts, cough remedies, treatments for fatigue, depression, sleeplessness, rheumatic pains, bathing sore eyes, toothache, headaches, stomach cramps, nervous disease, piles, and delirium tremens. For example a hundred poppy capsules could be bought for one shilling in 1800 – Pierce, M., *London Medical Review*, vol 3, no. X1V, 1800, p108, Current Prices of Drugs in the London Market.

The symptoms of poisoning by opium include giddiness, stupor, slow pulse, shallow breathing, contracted pupils, and insensibility. Opium was the poison which most claimed the attention of the physician and the medical jurist in the Victorian period. During the nineteenth century it was a medicine that was frequently used in all levels of society.⁴ As Elizabeth Lomax has commented, concerns for public health which prompted the Pharmacy Act of 1868 were often animated by cases of addiction and poisoning among the adult population, and poisoning due to dosing of children with opiate draughts⁵. The Pharmacy Act did not, however, exclude patent medicines containing opium and its derivatives which could be bought easily in any apothecary, grocers, general dealers, or even small corner shops.⁶

Throughout the nineteenth and early twentieth centuries children were often killed, deliberately or accidentally, with opium preparations.⁷ Record and discussion of these cases has not been profuse despite their abundance. Physicians of the period very frequently regarded various symptoms in children as the result of general problems such as congestion, inflammation, or of a derangement of a balance between the

⁴ The history of the medicinal use of opium extends at least from the third century BC when Theophrastus referred to its use under the name of meconium. In the next three centuries the cultivation of the opium poppy spread to the Mediterranean countries and was later extended to Persia, India and China by the Arab traders – Polson, C.J., and Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London, 1973, p535.

⁵ Lomax, E., ‘The Uses and Abuses of Opiates in Nineteenth-Century England,’ *Bulletin of the History of Medicine* 47 (1973), 167-76.

⁶ Thus, although Schedule A, Part 2 of the 1868 Act provided that opium and all preparations of opium or of poppies could only be sold by duly registered pharmaceutical chemists, and that such sales had to be duly recorded in the form set forth in Schedule F to the 1868 Act, patent medicines were excluded. Indeed, it was not until the passing of the Dangerous Drugs Act in 1920 that it became illegal to possess opiates without a doctor’s prescription. For “professional” opposition to patent medicines, see Petition from Royal College of Physicians, Edinburgh to House of Commons ----Thomson, A.T., *Lancet*, vol 1, 1859, p294 Professional Opposition to Patent Medicines.

⁷ “We almost daily see infants in this city poisoned by opium, but as this arises from the general practice of exhibiting “sleeping draughts” by nurses, and as these are quack nostrums, we can seldom discover the exact quantity that has been given, more especially as the publicans prepare their diacodium, and the druggists their composing cordials of very different strengths.” - Ryan, M., *London Medical and Surgical Journal*, vol 7, 1831, p148, Homicide by Poisoning.

venous and arterial systems.⁸ Further, as has been shown in the preceding chapters, the science of toxicology could only just keep pace with the poisoners and, in particular, the detection of opium and its derivatives was often difficult.⁹ In these ways, a profusion of murders are likely to have occurred which were never detected. Moreover, the overall patterning of cases in Scotland, has not been fully elaborated.

5.2 Profile of Opium Murder Cases

Research has disclosed twelve opium poisoning cases, 19% of all cases, within the chosen time period. This is a figure which, given the ubiquity of opiates in Victorian society seems low in comparison to the number of arsenic poisoning cases although nine (75%) of the opium cases occurred in the Victorian era. Figures for English criminal cases show that there were fifty-two cases between 1750-1914 representing 15% of cases and that at the Old Bailey between 1750 and 1914 there were ten opium poisoning cases representing 21% of cases.¹⁰ The earliest cases in this work address the death of adults, but the majority of cases, occurring in the mid-Victorian period, concern the death of children. Naturally the widespread availability of opium preparations led to a great potential for their misuse, particularly given the addictive properties of the drug.¹¹ Further, opium itself developed attractive social symbolic associations with mysticism due to its connections to China and the Islamic world and with decadent bohemianism due to the writings of intellectuals like de Quincey. It is possible, if exact figures for use and abuse of opium products had existed, that it

⁸ Taylor, Alfred, Swaine., *EMJ*, vol 14, 1868-1869, pp744-748, The Principles & Practice of Medical Jurisprudence.

⁹ "It may be laid down therefore as a general rule that in poisoning with opium, the medical jurist, by the methods of analysis yet known, will often fail in procuring satisfactory evidence, and sometimes fail to obtain any evidence at all, of the existence of the poison in the contents of the stomach." – Per Dr Christison – *London Medical and Surgical Journal*, vol 7, 1831, p149, Homicide by Poisoning.

¹⁰ Watson, K., *Poisoned Lives: English Poisoners and their Victims*, Hambledon and London, London, 2004, p33; Forbes, T.R., *Surgeons at the Old Bailey*, Yale University Press, London, 1985, Table 8.

¹¹ See Foochow, C., *EMJ*, vol XVII, 1872-1873, pp478-479, Opium Smoking; Myers, W.H., *EMJ*, vol XXXI, 1886, pp357-358, Extraordinary Case of Opium Eating Cured; De Quincey, Thomas., *Confessions of an English Opium Eater*, London, 1821, reprinted Penguin Books, London, 1997.

could be comparable to the modern day employment and misuse of heroin, methadone and benzodiazepines.¹²

There were, and are, no means of detecting opium except by its smell and other physical properties.¹³ Analysis of suspected poisoning by opium was, therefore, limited to the detection of morphine and other alkaloids. Hence the detection of opium posed many problems for the toxicologist and indeed, Taylor, the renowned forensic toxicologist of the nineteenth century, was not convinced that any of the contemporaneous tests for opium were entirely unambiguous.¹⁴

Many of the criteria used to diagnose poisoning by opium cannot, therefore, be taken as reliable for chemical analysis. The temptation to poison must have been great for those at the lower end of the social spectrum, where life was racked with poverty, disease, malnutrition, poor sanitation and hygiene, squalid living conditions and financial deprivation and exploitation. Indeed, in an era where people were so vulnerable to illness, and the prescribing of regular medicine often lacking or misguided, it is of little surprise that children and the weak were easy prey to those with an inclination to abuse the potentially poisonous properties of opiate preparations. The account of the cases is thus divided thus into two sections. Firstly, I deal with the deaths of adults, wherein the primary motive was pecuniary gain and this was achieved either by deliberate murder or by attempts to drug which unwittingly resulted in death. Secondly I deal with cases involving the deaths of children wherein the administration of opiate preparations caused death either intentionally or unintentionally.

¹² Like opium products heroin, methadone and benzodiazepines all influence mood by lessening emotional distress and producing a tranquil pleasantness which for many is positively enjoyable. There are many modern benzodiazepines in use, but the most common are probably valium, diazepam and lorazepam.

¹³ Natural opium is a complex mixture of substances, mainly morphine and meconic acid.

¹⁴ Coley, N.G., *Medical History*, vol 35, 1991, p424, Alfred Swaine Taylor MD, FRS, (1806-1880).

5.3 Cases of Opium Poisoning of Adults

The first case in this sequence for poisoning with opium occurred in Glasgow in 1800. A certain Mr James Lockhart and his maid-servant, Betty Forbes, were accused of murdering Lockhart's former wife, Marion Gardner by putting opium into ale. Whilst it was clearly proved that Lockhart had purchased great quantities of opium, Marion's body was not opened until a fortnight after death and no appearances of poison were noted. On the want of any evidence as to the *corpus delicti*, the Court directed the jury to acquit Lockhart and Forbes.¹⁵

In 1828, a year notable in history for the serial murders carried out in Edinburgh by Burke and Hare,¹⁶ a second opium poisoning case is also noted in the Records of the Lord Advocate. This case involved the conviction of a couple who had committed murder by poison for profit. On 15th December 1828, a blacksmith and his wife, John and Catherine Stuart, boarded the steamer, the Toward Castle, travelling from Loch Fyne to Glasgow.¹⁷ On board the couple started drinking with a fellow passenger, Robert Lamont, in a downstairs saloon. From the evidence of the steward, John McNairn, the Stuarts and Lamont drank "three gills of whisky, three bottles of porter and a dozen of ale."¹⁸

When the steamer reached Renfrew Ferry, Robert Lamont's cousin, John, who had been travelling with him, went below to summon him from the saloon. He found Robert lying on the floor, insensible, and with his empty pocket book lying nearby. The Captain was at once informed that a robbery had taken place, the Stuarts were

¹⁵ See Burnett, J.A., *A Treatise on Various Branches of the Criminal Law of Scotland*, George Ramsay & Co., London, 1811, p549.

¹⁶ Between December 1827 and October 1828 Burke and Hare were responsible for the murder of sixteen persons by physical violence, whose bodies they sold to Dr Knox at Surgeon's Hall for £8-£10 each.

¹⁷ The Toward Castle was a wooden steamer ship of some 79 tons and 45 horse-power which sailed between Glasgow and Loch Fyne. The couple boarded at Loch Fyne, on her return run to Glasgow from Inverary.

¹⁸ Record of the Lord Advocate AD14/29/108. It was noticed that before Robert Lamont drank, Mrs Stuart "put the tumbler in below her mantle, and once, as her husband was about to drink, she pulled the tumbler from his mouth and spilt the contents over his breast and he damned her for it."

taken and searched, a black purse and £19.7.0 being found; all of these were later proved to be the property of Robert Lamont.

When the ship reached the Broomielaw at six o'clock in the evening, Stuart and his wife were taken into custody. A further search revealed two empty laudanum bottles in their possession.¹⁹ A doctor, called to attend Robert Lamont, was of opinion that he exhibited many of the symptoms of laudanum poisoning.²⁰ A stomach pump was, therefore, applied. Lamont, however, never regained consciousness and died that evening aboard the ship. A post-mortem was carried out on 16th December by Dr James Corkindale and Dr Fleming of Glasgow, which revealed characteristics of laudanum poisoning.²¹ Fluid taken from the stomach was subjected to various chemical tests by Dr Andrew Ure and Dr James Corkindale of Glasgow. Although no details of any chemical tests are given in the records, both doctors stated that they had determined the presence of opium or its spirit solution, laudanum, in the fluid from the stomach.

On Tuesday, 14th July 1829, the High Court of Justiciary in Edinburgh sat for the trial of the Stuarts.²² During the trial all principal circumstances for establishing guilt were proved.²³ The motive of theft was prominently noted. In particular, it was proved that the panel Stuart had no money when he went on board the boat, but that soon afterwards was in possession of a sum, not only similar in amount, but consisting of the same description of bank notes of which Lamont had been robbed.²⁴ A further circumstance of corroboration was the black purse, found in the possession of John Stuart, which was proved to have belonged to the deceased.²⁵ In addition, evidence

¹⁹ Thus proving principal circumstance of proof of possession of poison.

²⁰ Appendix 18, symptoms -: 2, 3, 4, 5, 6, 7, 8, 11, 17.

²¹ Appendix 19, appearances -: 1, 2.

²² It is of interest is that the indictment did not libel the place of death. Indeed, no place of death was set forth at all, and despite objections from the defence, the indictment was found to be relevant.

²³ Possession of poison by the accused, secret administration, and motive.

²⁴ The Master of the Toward Castle, William Stewart, recalled how Stuart and his wife pled poverty when he tried to collect their fare. The couple claimed they had lost their passage money, but did eventually pay with silver. William Stewart, however, saw neither a black purse nor notes.

²⁵ His cousin saw him with the purse on the voyage and his daughter recognised the purse as her own sewing.

was given at trial by two of the Stuarts' fellow prisoners in the Tolbooth in Edinburgh.²⁶ Both deponents attested that John Stuart had confessed to the crime, thus corroborating each other's statement. The facts were thus clearly established. No witnesses were called for the defence and after an absence of only five minutes the jury returned a unanimous verdict of 'Guilty' against both panels, upon all the charges against them.²⁷ Both were sentenced to be hanged in Edinburgh on 19th August 1829.²⁸

Prior to the trial, the couple fully acknowledged their guilt to their own counsel. In addition, Stuart confessed that he was aware of no less than seven others having been murdered through the administration of laudanum by himself and his wife.²⁹ It would seem, therefore, that this couple had systematically adopted "drugging" as a means of livelihood. No true figure can be put upon their murderous activities, let alone the number of people whose lives they had put at risk through "drugging". It would seem likely that the activities of the Stuarts alone would substantially swell the records of poisoning by opium had their crimes been detected.³⁰

²⁶ Alexander Malcolm Logan and Archibald Anderson, both in prison for the crime of theft.

²⁷ The panels had been accused of the common law charge of the administration of laudanum to any of the lieges to the injury of the person, and also of the statutory charge of administration with intent to murder or disable, murder and theft.

²⁸ Interestingly, while the hanging of Burke on January 28th, 1829, drew a crowd of 25,000, this double hanging attracted only a crowd of some 10,000.

²⁹ The murder of a gentleman in a tavern in Glasgow; the murder of a ferryman in Kirkcudbright; the murder of a gentleman in Bridgegate, Glasgow; the murder of a gentleman in a public house in Princes Street, Edinburgh. This testimony was also borne out by the testimony of the two fellow prisoners, Alexander Logan and Archibald Alison. Stuart had told them that he had been giving laudanum to people all over the country, then robbing them and was certain such persons had all died. Record of the Lord Advocate AD14/29/108.

³⁰ One of the ballads sold at the gallows-foot for a penny, was as follows :- "A certain man upon a plan he put us both one day, How we could raise money with ease and no lives take away, By giving laudanum to them and putting them to sleep, So by an oath he bound us both the secret for to keep. In the Bridgegate of Glasgow once this horrid scheme we try'd. The dose being strong, it was not long before the poor man dy'd; Of the same death, in the Trongate, another dy'd also; We left the place to shun disgrace, to Ireland we did go. We never thought we would be brought to trial for this crime."

The practice of “drugging” with opium appears to have been not uncommon in the early 1800s aided, no doubt, by the ease with which opium could be obtained. The third case in the sequence is the related trial of James and Mary Byers in 1831 for the murder of John Martin, which corroborates the spread of the pattern of behaviour which the Stuarts displayed. On March 9th 1831, husband and wife, James and Mary Byers, arrived in Glasgow from Belfast. Accompanying them was an elderly uncle, John Martin, and Robert Byers, a brother of James. The party took lodgings in Steel Street, Glasgow. John Martin appears to have been a man of some means.³¹ On the forenoon of March 10th, James and Mary Byers, together with another lodger, Agnes Cairnie, as well as John Martin, entered the shop of James Rennie, publican, in the High Street. There, according to Agnes, the couple plied Martin with porter from a tumbler. As a consequence he quickly fell into something akin to a stupor and required assistance back to the lodgings.³²

In the early evening a surgeon, Edward Dillon, called to see Martin, having earlier that morning been visited by him.³³ Finding Martin drowsy and stupid, however, and fearing he might be robbed, Dillon decided to move Martin to his own lodgings.³⁴ There Martin remained insensible. His suspicions raised, Dillon examined Martin’s pocket book and found most of his money gone. Suspecting Martin had been poisoned, Dillon called another doctor, Dr Nielson, and the police were advised. Martin died just before 6am on the 11th March with the result that both James and Mary Byers were apprehended and charged with murder and theft.³⁵

During the trial, corroborated evidence was given by Dr Edward Dillon and Dr James Corkindale, that Martin had displayed symptoms that resembled those of poisoning

³¹ He had in his possession a hundred pound bond, a promissory note for £6.10/-, a £30 bank of Ireland note, a cheque for £70, four small bank notes and some silver change.

³² To be noted is that Martin only drank from the tumbler Mary Byers gave him, and that nobody else drank from that tumbler – Record of the Lord Advocate AD14/31/268.

³³ This was in relation to a letter which Martin had brought from Dillon’s father in Ireland.

³⁴ So much was Martin under the influence of something, however, that he required much support to reach the new lodgings.

³⁵ Mary Byers was found in Glasgow, but James had managed to make his way back to Belfast, where he was apprehended and brought back to Glasgow.

with laudanum.³⁶ The same doctors also carried out a post-mortem. This did not enable them to account with definite proof for the sudden death, but they were still of opinion that death had been due to laudanum poisoning. At trial, the plain inference was that Martin had died from the administration of laudanum. With regards to possession of poison, evidence was led from John Lamont, shop assistant, that on Tuesday 10th March, Mary Byers had purchased twopence worth of laudanum from the shop where he worked in the Candleriggs in Glasgow. Also evidence was given by Alexander Milson, who stated that on the same day, Mary had purchased a further penny worth of laudanum from his brother's chemist shop in the High Street, Glasgow.³⁷

Thus was proof of possession established, but motive also had to be considered at the trial. Motive was clearly attributable to the couple's desire for pecuniary gain. In particular, they had received change for a thirty-shilling Bank of Ireland note from Patrick Sandlin, publican in Bridgegate Street, which was proved to have originally belonged to Martin. Further, when Mary Byers was searched, she was found in possession of James Martin's hundred pound bond, plus other monies alleged to have belonged to him.

At the conclusion of the trial the jury unanimously found the pair guilty, and they were sentenced to be hanged. Indeed, after her condemnation, Mary confessed to purchasing and administering the laudanum and taking four thirty-shilling Irish banknotes from Martin's pockets. However, much as in the case relating to the Stuarts, murderous intent was most likely secondary to the desire simply to drug and rob Martin. Mary Byers denied any explicit intention to murder.³⁸ James Byers claimed innocence to the end, denying any knowledge of the plan.

Lastly in this section is the trial of Eugène Marie Chantrelle, which is the final case in this chapter to concern the murder of an adult. Though the case is treated here, it is the tenth opium trial, chronologically speaking, and took place in 1878. Although

³⁶ Appendix 18, symptoms :- 2, 4, 5, 6, 7, 8, 10, 11, 17, 19, 21.

³⁷ Record of the Lord Advocate AD14/31/268.

³⁸ Young, A.F., *The Scottish Encyclopaedia of Scottish Executions 1750 to 1963*, Eric Dobby Publishing Ltd, Kent, 1998, p106.

Chantrelle's trial for the murder of his wife is of great interest and importance, it already occupies a prominent position in the history of poisoning, having been recorded and written about extensively in Scottish criminal jurisprudence.³⁹ The dominant feature of this case was the fact that Chantrelle had gone to great effort to make it appear that his wife had died from an accidental escape of gas in her bedroom, whereas he had in fact poisoned her with opium.⁴⁰ Samples of fluid taken from the stomach of Chantrelle's wife, Elizabeth, following death showed that she had swallowed a substantial amount of extract of opium.

On October 18th, 1877, Chantrelle had insured the life of his spouse for £1,000, the policy being so framed that the insurance would only be paid if her death was caused by accident. In evidence given by a witness from the insurance company, it was stated that the accused had intimated the desire for the policy to cover only fatal accident.⁴¹ That Chantrelle ever had any affection for his wife is doubtful in the light of his conduct towards her. He frequently abused her without reason, was violent towards her, threatened to poison her, and to her knowledge was systematically unfaithful to her.⁴² The only motive thus for his insuring his wife's life was financial gain, money of which Chantrelle felt himself much in need. Despite protestations of innocence which he never recounted, medical evidence, proof of possession, motive and Chantrelle's character counted against him. He was found guilty and hanged.

5.4 Cases of Opium Poisoning of Children

Just as in the cases of infant poisoning with corrosive acids, the grim plight of Victorian children and in particular unwanted children amongst the Victorian poor led to motives for poisoning. Two kinds of child poisoning are in evidence in this section. First are cases where, just as in the trial of Elizabeth Walker for the poisoning of her

³⁹ In particular see Smith, Duncan, A., *Trial of Eugène Marie Chantrelle Noteable Scottish Trials*, W Hodge & Co Ltd, Glasgow & Edinburgh, 1906; Glaister, John., *The Power of Poison*, Christopher Johnson, London, 1954, pp183-192.

⁴⁰ Glaister, John., *The Power of Poison*, Christopher Johnson, London, 1954, p183.

⁴¹ Ibid, p184.

⁴² Smith, Duncan., *Trial of Eugène Marie Chantrelle*, Notable Scottish Trials, W Hodge & Co Ltd, Glasgow & Edinburgh, 1906, pp3-4.

son with nitre in Chapter Three, mothers in financial difficulty could often simply not afford to keep children, and in desperation, were willing to countenance murder. During the 19th and early 20th centuries respectable society offered no support and was often openly hostile to the large number of children born out of wedlock and to their mothers. These children were often born to women working in domestic service, who could not, under their conditions of employment, keep the child. Indeed, this scenario was all too common, and the culture, the oppressive poverty and the economic realities of working-class life often led to fatal measures. Secondly, cases are discussed which concern nursemaids whose motivations for murder with poison are often more diverse and difficult to discover.

Although there are only eight cases for the poisoning of children with opium, it seems likely that this figure represents only a fraction of children poisoned by opium products.⁴³ Indeed, it is likely that for every case that was exposed, tens or even hundreds of others went undetected. As Henry Littlejohn wrote in the British Medical Journal in 1868, “there is not the slightest difficulty in disposing of any number of children, so that they may never give any further trouble and never be heard of.”⁴⁴

The first case in this section concerns Jean Crawford, a domestic servant in Saltcoats, who, on the 4th October 1847, administered sixty drops of laudanum to her illegitimate fifteen month old son.⁴⁵ The child soon fell into a stupor and died within a few hours.⁴⁶ During the trial on December 6th 1847, the panel pleaded guilty to the charge of culpable homicide and was sentenced to one year in prison. Notable in this case is the lenient sentence passed. Indeed, in the remaining cases in this chapter there

⁴³ Often desperate parents would murder their children to enable them to collect a small sum of burial money with which to keep themselves alive – Altick, R.D., *Victorian Studies in Scarlet*, L M Dent & Sons Ltd, 1972, p285.

⁴⁴ Littlejohn, Henry, Duncan, – *BMJ*, vol 3, 1868, p303, The Doping of Infants.

⁴⁵ Advocate H.M. v Crawford (1847) Arkley, p395. Note that according to Henry D Littlejohn, forensic toxicologist, six to eight drops would be sufficient to kill a child, and that because of the lawful widespread use of opium products most mothers would have been aware of this- Record of the Lord Advocate AD14/73/217.

⁴⁶ Appendix 18, symptoms :- 3, 4, 6, 7, 11, 17, 23.

also appears to have been a very lenient administration of justice consonant perhaps with sympathy for the straits in which these women found themselves.

In the second case of the death of a child twenty-seven year old Lydia Dodds of King Street, Glasgow, made three separate purchases of laudanum from different druggists in the Saltmarket, Glasgow, on Tuesday 7th July 1868. The last purchase was made at around five thirty in the afternoon.⁴⁷ Lydia was then seen around six o'clock sitting with her daughter in a close in the Saltmarket by neighbours Mary Tierney and Mary Russell. Lydia was very much under the influence of alcohol and told both women that her husband had left her for someone else and that she could now not afford to keep her child. Lydia then put a cup to the child's mouth and poured in a brown liquid following which she threw the cup away and it smashed into pieces.⁴⁸

A passing constable, Donald M'Kellar, approached the close where Lydia was sitting and crying. She informed him that she had taken four half pennies worth of laudanum herself in the hope of dying and given a halfpenny worth to the child. Indeed, on being taken to the local police station Dodds admitted that she had intended to poison her daughter. Although the child recovered, the symptoms observed by a Dr MacGill of slow pulse, slow respiration, moist skin and contracted pupils were all indicative of laudanum poisoning.⁴⁹ Lydia was charged with administering poison with intent to kill. During her trial all the principal circumstances for establishing guilt were proved.⁵⁰ In addition, there was direct testimony from Mary Tierney, corroborated by Mary Russell, as to their observation of the administration of the poison.⁵¹ Lydia pleaded guilty to the common law charge of administering poison. She was sentenced to six calendar months imprisonment.

⁴⁷ This last purchase of laudanum was made from Druggist Robert Whitehead, 38 Saltmarket Street, Glasgow. Here Lydia purchased a half penny worth of laudanum (60 drops) –Record of the Lord Advocate AD14/68/131.

⁴⁸ Record of the Lord Advocate AD14/68/131.

⁴⁹ Appendix 18, symptoms -; 2, 3, 6, 8.

⁵⁰ Possession of poison, administration, motive.

⁵¹ Such testimony is very rare in poisoning cases. See Arsenic chapter, note 61.

In a further case, on the 26th February 1873, twenty-three year old Margaret Welsh administered an excess of laudanum to her seven month old son.⁵² Margaret, unmarried, worked as a laundress in a brothel in Edinburgh and, being, a single parent the child was a hindrance to her.⁵³ She therefore had to pay a certain Jane Robertson for child care.⁵⁴ On 25th February, Margaret was dismissed from her job. She arrived at the Robertson house the following day, stating that she would take the child away next morning. Margaret then went out to purchase whisky, which she and Jane drank. She then gave Jane money to go and buy cheese, and on her return told of how, in Jane's absence, she had given the child half a glass of whisky to put him to sleep. Margaret then proceeded to relate to Jane of how she had often thought of taking the child's life.⁵⁵ When Margaret left, Jane and her mother went to bed. However, on awakening the next morning they found the child dead.

Margaret returned to the house that morning and, on being asked what she had done, and shown an empty phial which the Robertson's had found in the fireplace, stated that she had given the child a penny worth of laudanum.⁵⁶ The police were called and Margaret arrested. Post-mortem examination and chemical tests carried out by Drs Henry Littlejohn and Douglas MacLagan revealed no trace of laudanum.⁵⁷ Such detection was, however, as already remarked, rarely successful in the nineteenth century.⁵⁸

With regards to the possession of poison by Margaret, a strong inference of guilt was created at trial. A witness, Alexander Ferrier, an apprentice druggist, stated that Margaret had purchased twopence worth of laudanum on Wednesday 20th February, from the shop where he worked in Nicolson Street. Indeed, the laudanum had been

⁵² Record of the Lord Advocate AD14/73/127.

⁵³ This was at 4 Hill Place, Edinburgh. Record of the Lord Advocate AD14/73/127.

⁵⁴ Jane Robertson lived with her mother at East Richmond Street, Edinburgh. For her services she was paid 5 shillings a week, the money being taken out of Margaret's wages every week by her employer.

⁵⁵ Record of the Lord Advocate AD14/73/127.

⁵⁶ Ibid.

⁵⁷ Appendix 17, tests -: A4, A5, B5.

⁵⁸ This was particularly the case with the fluid preparations of opium (such as laudanum) which, from their rapid absorption and transformation within the system, were in most instances rendered incapable of detection by chemical means.

put into a phial identical to the one found in the fireplace in the Robertson home.⁵⁹ Further, Margaret had ample opportunity to administer laudanum to her child when Jane went out to purchase cheese, and as regards motive, the court considered not only the fact that Margaret had lost her job, but many statements given by witnesses who had heard Margaret say she wished her child dead.⁶⁰ Since the above was all proved, clear evidence of guilt was established. In fact, Margaret eventually pleaded guilty to her crime at trial and was sentenced to twelve months imprisonment.

In a case from the 18th December 1872, where fewer details are extant, Catherine Findlay was charged with culpable homicide. This followed the administration of ten drops of laudanum mixed in water and milk to her ten month old daughter, Ann, whilst in the High Street in Kincardine, Perth.⁶¹ The laudanum was given to the child directly from a teaspoon and at trial Catherine stated that she had given the child the laudanum as a sleeping aid and was in the habit of doing this. Indeed, Catherine frequently sent her eight year old daughter to purchase a penny worth of laudanum from the local druggist, Dr Erskine. The infant died the same day and at trial Catherine pleaded guilty and was sentenced to fifteen months imprisonment.⁶²

These cases establish clear patterns of the use and abuse of opiates by mothers in the nineteenth century. As in the Findlay case, mothers living difficult and parlous existences commonly administered opiates simply to make child care easier and to lessen the demands of small children on their time and energy. In many cases, this risky practice is likely to have led to deaths akin to the case above. The desperation of crushing poverty also drove women to wish their child's death and even their own and opiates were commonly employed in such cases. Despite the lack of medical proof often available (certainly there was much less certainty in the case of opiates than in the case of arsenic, for instance) guilty verdicts were often passed. This can be attributed to the fact that many of the women committing these crimes were not hardened, devious criminals, but simply desperate. Hence, most pleaded guilty and

⁵⁹ Although perhaps not wholly reliable evidence for identification purposes, the phial was identified by the marking "York Glass" on its base.

⁶⁰ Record of the Lord Advocate AD14/73/127.

⁶¹ Record of the Lord Advocate AD14/73/258.

⁶² Justiciary Court Records JC11/106.

admitted to crimes which were borne more from despair than malice. Thus, as argued in earlier chapters, the circumstantial facts of the case were often seen to be more important to juries than medical testimony.

The second tranche of cases relates to administration of poison by nursemaids and servants. Throughout the nineteenth century and indeed until the First World War domestic service constituted the largest single employment for women Quite apart from housemaids and kitchen maids, many households employed nursery maids to care for their children. The practice of nursery maids and working mothers dosing infants with soothing syrups based on opium was a common reality in the past.⁶³ Medical officers believed that one of the major causes of infant mortality was the widespread practice of giving children opium products to quieten them and there were concerns about unqualified child doping by nursery maids. Indeed, the question was often brought up in Parliamentary debates and written about in medical press.⁶⁴ In fact, despite Alfred Swaine Taylor, the eminent forensic toxicologist, giving extensive evidence of infant mortality from opium to support his demand for the restriction of its sale and use, his campaign against child drugging was, both for cultural and economic reasons, ignored.⁶⁵ Indeed, with the practice of infant drugging being so commonplace and socially acceptable, it is of little surprise that so few cases of infant poisoning came to the attention of the authorities.

⁶³ Godfreys' was the most famous of those soothing syrups. A pharmacist remembered the days when he used to make up Godfreys' by the gallon: "I ----- can see with memorys' eye the fluted green syrup bottle with its recessed label in red and gold in which it was kept. I can smell still the oil of sassafras which, with alcohol and laudanum I stirred into the black treacle." – Galen, B., *Pharmaceutical Journal*, vol 10, 3rd series, 1879-1880, pp746-747, Poisoning by Godfrey's Cordial.

⁶⁴ See P.P. 1857, X11, (sess-2): Report from the Select Committee of the House of Lords on the Sale of Poisons etc Bill, 9-1852; P.P. 1871, V11, Select Committee on the Protection of Infant Life; P.P. 1867, XV11: Annual Report of the Registrar General, (London, HMSO), 1867; Galen, B., *Pharmaceutical Journal*, vol 6, 3rd series, 1875-1876, pp176-179, Alleged Child Poisoning; Galen, B., *Pharmaceutical Journal*, vol 10, 3rd series, 1879-1880, pp746-747, Poisoning by Godfrey's Cordial; Hall, M., *EMJ*, vol 12, 1816, pp423-424, The Effects of the Habit of Giving Opiates on the Infantine Constitution.

⁶⁵ P.P. 1857, X11, (sess-2), Report from the Select Committee of the House of Lords on the Sale of Poisons etc Bill, 9-1852; On Poisons in Relation to Medical Jurisprudence and Medicine, pp586-587.

On the 7th May 1857 Elizabeth Hamilton, a nursery maid, was charged with the crime of culpable homicide.⁶⁶ Elizabeth had been employed at a house in Grove Street, Edinburgh by Andrew Drysdale, a cashier at the Caledonian Distillery, and had administered ten drops of laudanum to three month old Andrew M'Neill Drysdale, who as a consequence, died on the 7th May 1857. Elizabeth pleaded guilty at trial and was sentenced to eighteen months imprisonment.

On the 14th April 1881 Agnes Leslie, a twenty-nine year old nursery maid in Edinburgh, was employed by William Henderson, Procurator Fiscal, to nurse his two month old son.⁶⁷ From the commencement of her employment the baby, Charles Henderson, grew pale and sickly, and his mother began to suspect that Agnes was drugging him. On May 6th 1881 Mrs Henderson entered the nursery around ten at night. There she found Agnes asleep in a chair and the infant with a pacifier, with tubing attached, inserted in its mouth. The child appeared to have difficulty breathing and she called her husband. On being told by Mr Henderson that the pacifier would probably be analysed Agnes produced a phial containing laudanum and said that she was entitled to administer it when she thought proper, as she was a certified nursery maid.⁶⁸

The family doctor, James Hunter, was called to attend the child. His opinion was that the infant was dangerously ill and that his general appearance and symptoms were consistent with the administration of an opiate.⁶⁹ In the early hours of May 7th the child died. Post-mortem examination carried out by Drs Douglas MacLagan and Henry Littlejohn, as in other cases above, failed to indicate the poisoning by laudanum which had been admitted. Further, chemical tests showed no evidence of laudanum having been administered.⁷⁰ During the trial of Agnes Leslie, both doctors emphasised the rarity of the chemical detection of laudanum in the stomach after death. In

⁶⁶ Advocate H.M. v Hamilton Irvine (1855-1857), pp738-739.

⁶⁷ This was, because his 3 year old son had scarlet fever and Mrs Henderson was unable to look after both children – Record of the Lord Advocate AD14/81/108.

⁶⁸ “I am a medical woman and am entitled to have it, and to give it to my patients when I think proper, but I never gave your child a drop of it.” - Record of the Lord Advocate AD14/81/108.

⁶⁹ Appendix 18, symptoms -: 3, 7, 8, 9, 10, 11, 21.

⁷⁰ Appendix 17, tests -: A5, B4, B5.

particular, Henry Littlejohn stated that he attributed much of the overall mortality among children to laudanum poisoning, from which both doctors strongly believed this child had died.⁷¹

Proof of possession of poison by Agnes was established at trial by the testimony of two Edinburgh doctors, John Halliday Groom and James Archibald Fidey. Both had frequently prescribed laudanum and chlorodyne for her to treat a uterine disease of a painful nature.⁷² Further, there was a tin box found in her possession containing bottles of ergot, laudanum, chlorodyne and chloroform liniment. Agnes Leslie had ample opportunity to administer laudanum to the infant. It is of interest that another child who had previously been in her care some years ago had died without any reasonable explanation.⁷³

There appears to have been no motive here for murder. Accordingly, the death of the child can only be attributed to Agnes Leslie's negligent and dangerous practice of drugging infants in her care, to quieten them, and her strong belief that she was entitled to administer laudanum to her charges, if she deemed it necessary.⁷⁴ At trial Agnes Leslie pleaded not guilty to murder and the jury unanimously found the libel 'Not Proven'.

⁷¹ "It is a well known fact in toxicology that comparatively low doses of laudanum (such as 10 teaspoonfuls) have not been detected after death." – Per Dr Henry D Littlejohn, Record of the Lord Advocate AD14/81/108.

⁷² Chlorodyne was most commonly associated with the name of Dr John Collis Brown who had first used the preparation while serving with the army in India. Chloroform and morphia were the main ingredients and the name chlorodyne made from the words "chloroform" and "anodyne" – See Brown, G., *Lancet*, vol 1, 1872, p72, Composition of Chlorodyne. Chlorodyne was recommended for coughs, colds, influenza, diarrhoea, stomach chills, colic, flatulence, bronchitis, croup, whooping cough, neuralgia and rheumatism.

⁷³ The death of this child was at the time attributed to the milk obtained from some Edinburgh dairy.

⁷⁴ Speculation gives rise to the question of how many other infants under the care of this woman had also been killed in this way. Interestingly, Dr Henry Littlejohn was of the firm opinion that no certified nurse should be entitled to give laudanum in any quantity, however small to an infant under any circumstances, without orders from a doctor.

At Glasgow, in August 1899, Kate Beattie, fifteen, a domestic servant, was tried with attempting to murder seventeen month old James Fairley. Laudanum was alleged to have been given to the child. Kate had initially been employed on wages of six shillings a month⁷⁵ by James Fairley, a machineman, and his wife Isabella. Her employment commenced on 2nd June 1899 and she was tasked to attend to their two children and undertake some light housework. Despite there having been no chemical tests made, evidence was given by Dr James Paterson to show that on 19th June 1899, the infant was suffering from very serious laudanum poisoning.⁷⁶ A statement from druggist's assistant Jane Stevenson that Kate had purchased twopence worth of laudanum on June 19th, went further to establishing her guilt.⁷⁷

Indeed, on being apprehended in Kelvinside Avenue on June 20th by Detective Officer Archibald McKenzie, and on being told that it was for "attempting to poison Mr Fairley's child", Kate replied "that's quite true; I don't know what made me do it".⁷⁸ In addition the court considered the statement of Cassie Devine, to whom Kate had recounted, in a moment of panic, how she had beaten the baby and then, thinking he was dying, purchased laudanum, administered it and then had run away.⁷⁹ Accordingly, it appeared conclusive that Kate had committed acts which fitted the description of attempted murder by poison. There was proof of possession of poison by Kate, and also opportunity for secret administration, but motive must also be considered. Motive was attributable to Kate's dissatisfaction with her job. Mrs Fairley had refused to pay Kate some of her wages in advance and Kate had stolen a gold watch, Albert chain, and a locket belonging to Mrs Fairley.⁸⁰ The facts of the case

⁷⁵ Record of the Lord Advocate AD14/99/67.

⁷⁶ Appendix 18, symptoms :- 3, 4, 6, 7, 8, 11, 17.

⁷⁷ Record of the Lord Advocate AD14/99/67.

⁷⁸ Ibid.

⁷⁹ Cassie Devine was a friend and had known Kate for about 4 months – Record of the Lord Advocate AD14/99/67.

⁸⁰ Mrs Fairley usually paid her servants on the last Saturday of every month. The Saturday before being her pay was due, Kate asked her employer for an advance of 3/3 to buy a skirt. Mrs Fairley refused and Kate was much annoyed at this.

were exceptionally well established.⁸¹ Kate, however, pleaded not guilty to attempted murder and the jury returned a surprising verdict of ‘Not Guilty’ most likely since there was motive for abuse and neglect of the child, but not attempted murder.⁸²

Another case occurred in 1862 involving a druggist’s apprentice, Charles Buchan. Buchan was charged with the crime of culpable homicide, in so far as he had held himself out as a competent person to prescribe a proper medicine for a child without enquiring about the age of the child or the state of the child’s health.⁸³ On the 19th November 1862 a certain John McAllister had attended at the shop of druggist, John Chalmers, in Stirling to obtain a bottle of cough medicine for his two year old son. Without making proper enquiries as to the age of the child, Charles Buchan prepared a mixture of one and a half ounces which contained at least an ounce of opium product along with a solution of morphia. The child died within a few hours of being given the first dose. At trial, however, counsel for the accused objected to the relevancy of the indictment in that it related how John McAllister intended to use the cough mixture when obtained, although no such facts had been communicated. The case was dismissed.

5.5 Conclusions

In the nineteenth and early twentieth centuries opiates were as readily available as aspirin is today, and the various symptoms of childish ailments could have been easily confused with those caused by the ingestion of opiates.⁸⁴ Medical opinions were also often erroneous. Moreover, opiates were often administered with no malicious intent

⁸¹ In particular, there was Kate’s statement to Detective Officer Archibald M’cKenzie, that she had poisoned the child. This evidence was corroborated by Mr Fairley, who had accompanied the Detective officer, in his search for Kate.

⁸² Justiciary Court Records JC/13/121.

⁸³ Advocate H. M v Buchan Irvine’s Justiciary Reports vol 1V (1861-1864), pp392-397.

⁸⁴ In the early 1800s East Indian opium could be purchased for 8 shillings and sixpence a pound- Pierce, M., *London Medical Review*, vol 3, March- June 1800, p108: Current Prices of Drugs in the London Market. Whilst in the mid 1800s, two drachms [7.40cc] could be bought for two pence- Forbes, T.R., *Surgeons at the Old Bailey*, Yale University Press, London, 1985, p151. A typical urban semi-skilled worker in 1841 paid 2d for a loaf of bread, 5d for a pound of meat and 9d for a pound of butter- Burnett, J.A., *A History of the Cost of Living*, Penguin, Middlesex, 1969, p23.

whatsoever and this clouded the possibility of charges of murder or attempted murder, particularly when post-mortem chemical testing could not be relied upon to prove when the doses administered had been excessive.

Prima facie, it would appear that the Victorians were sympathetic towards female criminals. Indeed, of twelve trials involving the poisoning of young children in the Victorian era in this thesis, ten (83%) involved women. Of note is that of these ten trials, seven (70%) involved the use of opium, two corrosive acids (20%) and one arsenic (10%). Further, although guilty verdicts were reached in six of these trials (60%) lenient sentences were given in all cases. Indeed, not once was the death penalty given, and only once was a capital crime charged. It is only fair to point out, though, that there was also a very lenient sentence given in the one case where a male was found guilty of murdering a child during this era.⁸⁵ This is also suggested in the chapter on poisoning by corrosive acids, where three of the trials for murder in the Victorian period resulted in two guilty verdicts, accompanied by very lenient sentences and one not proven verdict.⁸⁶

Culpable homicide was charged in seven (58%) of the twelve opium cases and of note is that women were involved in nine (75%) of these cases. Further, seven (58%) of the twelve cases involved women and children. Illegitimate pregnancy placed women in a desperate position in the past and burial societies provided an appalling opportunity for financial gain. Many young children were nothing more than an encumbrance. It required no more than common prudence and tolerable competence to poison a child successfully. Such a job was, therefore, not too difficult and the prospect of discovery sufficiently remote to make the risk worth taking.⁸⁷ In the case of nursemaids and domestics, most of the cases betray professional malpractice and negligence, and these aspects of malpractice were exacerbated by frustration at conditions of

⁸⁵ This was the case of Stewart Ogilvie in 1869. See Corrosive Acids chapter.

⁸⁶ See Corrosive Acid chapter. In particular the cases of Margaret Macdonald in 1859, Stewart Ogilvie in 1869 and Elizabeth Walker in 1884. These cases are compared with that of Barbara Malcolm, who in 1808 murdered her child with vitriol, was found guilty and executed.

⁸⁷ No death certification was required in Scotland until the passing of the Births, Deaths & Marriages (Scotland) Acts 1854-1860 in 1854. Also as most of the poor died far from the reach of the medical profession, only if doubt were already entertained were questions likely to be officially asked.

employment. Despite strong circumstantial evidence, I can find no records of convictions for the capital charge of child murder with opium.

In general, the courts' handling of the medical evidence in such cases demonstrated a reluctance to accept any evidence as conclusive when the life of an accused woman was in the balance. Indeed, it would appear in these cases that there was increased pressure on medical experts to provide the courts with certainty. Under this pressure, in opium cases, existing standards of medical knowledge, and inferences from the conduct of post-mortem examinations of infants were increasingly found wanting in comparison to trials for murder and attempted murder with other poisons such as arsenic, corrosive acids and strychnine. Thus, with apparent widespread opposition to the death penalty for child murder by women, it is of little surprise that seven out of eight (88%) of the cases in this chapter involving children, resulted in the reduced charge of culpable homicide.⁸⁸ In addition, although there were clear guilty verdicts in five (63%) of said cases, the average sentence for such a crime was only thirteen months.

There seems, thus, to have been a de facto substitution of very light penal servitude for the death penalty in such cases. While the recognition of the desperate existence of the mothers in the cases above is attributable to a Victorian sense of fair 'humanity' in sentencing, it also belies a certain social tolerance of the practices of the day of ridding oneself of unwanted children. Indeed, as Littlejohn stated during the trial of Agnes Leslie, a great amount of the mortality among children might have been attributable to doping with opium.⁸⁹ As Adler and Polk have argued, however, in consideration of many types of child murder by women in desperate circumstances, such cases are usually the result of careful if distressed planning, and are not the result

⁸⁸ However, jurists, the press, and at least a segment of the medical profession in the 1860s and 1870s were disturbed by the fact that such trials almost never resulted in a conviction for murder. See Ryan, W.B., *Infanticide, its Law, Prevalence and History*, Churchill, London, 1962, pp48-62; Langer, W.L., *History of Childhood*, 1:353, 1974, p66, Infanticide: a Historical Survey.

⁸⁹ "I think that I have been able to trace among the lower orders in Edinburgh the effects of such a system of drugging with opium. It leads to early death and should the infant survive its first effects there can be no doubt that the development of the child is interfered with -----." – Record of the Lord Advocate AD14/81/108.

of an emotional loss of control. They write that such responses are the result of: “sheer frustration in light of...overall circumstances...they are taking the only action that is left to them to assure the happiness of their children.”⁹⁰ That Victorian society did little to prevent the lives of the poor from reaching such desperation is the cause of the plethora of such cases.

I submit on the other hand, that available records are not extensive enough to embrace the true significance of the murder of adults by opium in the past. In particular, laudanum was often used to assist in the conventional crime of theft, by pouring it into the drinks of unsuspecting victims. The modern equivalent exists today in the form of rohypnol which is an extremely fast acting benzodiazepine inducing sleep and commonly known today as “the date rape drug”. Such adulteration of alcohol could quite easily lead to death and there was a notable ineptitude amongst physicians in their failure to detect laudanum. It is highly likely therefore, that, whether murder was premeditated or not, a great many people more than records show, died from the criminal abuse of opiates in the nineteenth century.

Since post-mortem examination struggled to prove when opiate dosage had been excessive, it remained difficult for science to separate malicious from accidental poisoning. Victorian society was conditioned to expect both commonplace medication and self-medication with opiates and also to expect the use of opiates to end life among those individuals in the direst of social straits. Thus, in contrast to the cases of arsenic poisoning for instance, wherein the development of chemical testing was central to alerting the government and the public to the widespread dangers of the widespread sale of the poison, in the case of opiates, juries had other, social and circumstantial methods of evaluating cases. Once again, science could not play a constructive role in cases where murder with opiates needed to be separated from culpable homicide and accidental death.

⁹⁰ Adler, C. and Polk, K., *Child Victims of Homicide*, Cambridge University Press, 2001, p65.

APPENDIX 16
OPIUM POISONING CASES 1800 - 1899

Date	Place	Accused	Victim(s)	Method of Administration	Excuse for Obtaining Laudanum	What Happened to Victim(s)	Trial Verdict
1800	Glasgow	Lockhart	Wife	Ale	Not Known	Died	Not Guilty
1828	Steamer- The Toward Castle	John & Catherine Stuart	Robert Lamont	Ale & Porter	Liver Complaint	Died	Guilty- Executed
1831	Glasgow	James & Mary Byers	John Martin	Porter	Not Known	Died	Guilty- Executed
1857	Saltcoats	Jean Crawford	Son- Matthew Crawford- 15mths	Direct from Teaspoon	Not Known	Died	Guilty - 12 Months
1857	Edinburgh	Elizabeth Hamilton- Nurse	Andrew Drysdale- 3mths	Direct from Teaspoon	Not Known	Died	Guilty - 18 Months
1863	Stirling	Charles Buchan	Andrew McAllister- 3mths	As Medicine	Pretending to be Assistant Druggist	Died	Technical Acquittal

APPENDIX 16 (CONTD)

Opium Poisoning Cases

Date	Place	Accused	Victim(s)	Method of Administration	Excuse for Obtaining Laudanum	What Happened to Victim(s)	Trial Verdict
1868	Glasgow	Lydia Dodds	Daughter- Janet Dodds-5 mths	Direct from Cup	Stomach Cramps	Died	Guilty – 6 Months
1873	Edinburgh	Margaret Welsh- Domestic Servant	Son- Charles Welsh-7 mths	In Whisky	Toothache	Died	Guilty - 12 Months
1873	Perth	Catherine Finlay	Daughter- Ann Finlay-10 mths	Direct from Teaspoon	As Sleeping Aid	Died	Guilty– 15 Months
1878	Edinburgh	Eugène Marie Chantrelle	Wife- Elizabeth Dyer	In Orange or Lemonade	For Dispensing as Pills	Died	Guilty - Executed
1881	Edinburgh	Agnes Leslie- Nurse	Charles Henderson- 2 mths	On Rubber Teat	Prescription Medicine	Died	Not Proven
1899	Edinburgh	Kate Beattie Domestic Servant	James Fairley – 17 mths	Direct from Teaspoon	For Mistress	Survived	Not Guilty

APPENDIX 17⁹¹

Historical Tests Employed For Detecting Opium Constituents⁹²

A. Morphine

1. Crystals melt on heating, becoming dark coloured; and burn like a resin evolving ammonia.
2. Crystals are sparingly soluble in cold water and will dissolve in 100 parts of boiling water to give a faintly alkaline solution.
3. When concentrated sulphuric acid is added to morphine crystals a pink colour is obtained. On the addition of potassium chromate solution, a green colour is obtained.
4. Dissolve suspect crystals in hydrochloric acid and then add concentrated nitric acid. A deep orange colour is obtained if morphine is present.
5. Morphine crystals plus concentrated nitric acid gives a deep orange-red solution plus nitrogen dioxide.
6. To suspect aqueous solution of morphine add saturated iron trichloride solution and neutralise with potassium hydroxide. An inky blue solution is obtained which is destroyed by the addition of hydrochloric acid if morphine is present.
7. To suspect aqueous solution add a cold iodic solution. If morphine is present a brown colour is obtained due to the presence of morphine and iodine.

⁹¹ **Note:** There were no means of detecting opium except by its smell and other physical properties. Analysis was, therefore limited to the detection of both morphine and meconic acid. (Taylor, Alfred, Swaine, *On Poisons in Relation to Medical Jurisprudence and Medicine*, 2nd edn, Churchill, New Burlington Street, London, 1859, p621)

⁹² Tests 1, 2, 3, 4, 6, 7, 9 – Taylor, Alfred, Swaine, *On Poisons in Relation to Medical Jurisprudence and Medicine*, 2nd edn, Churchill, New Burlington Street, London, 1859, pp621-622.

Tests 5, 8 – Christison, Robert, *A Treatise on Poisons in Relation to Medical Jurisprudence, Physiology and the Practice of Physic*, Adam Black & Longman, Rees, Orme, Brown & Green, London, 1829, p517 -519.

8. If the suspected matter is solid wash with distilled water acidulated with acetic acid. If fluid, dilute with same. The solution having been warmed filtered and evaporated to the consistency of thin syrup the animal matter is to be separated by treating the residue with boiling alcohol. To the alcoholic solution previously filtered, add subacetate of lead as long as it causes precipitation then filter and wash. Treat the fluid part with sulphuretted hydrogen to throw down any lead which may remain in solution. It is then to be filtered while cold, and evaporated sufficiently in a vapour- bath. If, notwithstanding the action of the salt of lead and that of the sulphuretted-hydrogen, the liquid is considerably coloured, the colour must be destroyed by filtering it through animal charcoal. The solution thus eventually procured can be subject to the tests for morphia as formerly mentioned.
9. Morphia crystals are fine quadrangular prisms, which are white.

B. Meconic Acid⁹³

1. Forms reddish crystals, which are sparingly soluble in water.
2. In dilute nitric acid gives a white precipitate with lead acetate and a deep red colour with ferric chloride, which can be destroyed by sulphur dioxide or stannous chloride, but not by corrosive sublimate or gold chloride.
3. Add lead acetate solution to suspect solution in dilute acetic acid solution and a yellow-white precipitate is obtained if meconic acid present.
4. To suspect solution add saturated iron trichloride solution and a deep red solution is obtained if meconic acid is present. On the addition of sulphur dioxide or tin chloride solution, the red colour is destroyed. On the addition of dilute hydrochloric acid, mercuric or gold chloride there will be no effect.
5. Meconic acid solution will give a pale green precipitate with the addition of sulphate of copper.

⁹³ **Note**

Tests 1, 2, 3, 4 – Taylor, Alfred, Swaine, *On Poisons in Relation to Medical Jurisprudence and Medicine*, 2nd edn, John Churchill, New Burlington Street, London, 1859, pp623-624.

Test 5 – Christison, Robert, *A Treatise on Poisons in Relation to Medical Jurisprudence, Physiology and the Practice of Physic*, Adam Black and Longman, Rees, Orme, Brown & Green, London, 1829, p517.

APPENDIX 18

Laudanum Poisoning Symptoms⁹⁴

- 1.** Stupor.
- 2.** Giddiness
- 3.** Slow Respiration.
- 4.** Insensibility to external impressions.
- 5.** Power of motion completely lost.
- 6.** Eyes closed or half-open.
- 7.** Pulse slow.
- 8.** Bronchial Irritation.
- 9.** Loud Breathing.
- 10.** Pale countenance.
- 11.** Comatose State.
- 12.** Unable to swallow.
- 13.** Initial feeling of well being.
- 14.** Headache.
- 15.** Weariness.
- 16.** Sense of weight in limbs.
- 17.** Skin moist and warm with perspiration.

94 Note

Symptoms 1, 2, 3, 4, 5, 6, 7 – Ryan, M, “Homicide by Poisoning”, *London Medical & Surgical Journal*, vol 7, 1831, p148.

Symptoms 9, 10, 11, 14, 15, 16, 17, 18, 19, 20, 21, 23 – Taylor, Alfred, Swaine, *On poisons in Relation to medical Jurisprudence and Medicine*, 2nd edn, John Churchill, New Burlington Street, 1859, pp587-591.

Symptoms 8, 12, 13, 22, 24 are taken from Records of The Lord Advocate.

These symptoms are listed in this appendix due to the difficulties faced in the chemical detection of opium and in particular due to the frequency with which children were dosed with ‘soothing syrups of laudanum’

- 18.** Pupils strongly contracted in early stages.
- 19.** Pupils insensitive to light.
- 20.** Nausea and vomiting.
- 21.** Falling of body temperature.
- 22.** May be postural hypertension.
- 23.** Breath may emit odour of opium.
- 24.** Muscles become relaxed.

APPENDIX 19

Post-Mortem Appearances⁹⁵

1. May be smell of opium in stomach contents.
2. Marked cyanotic lividity of both the skin and the organs.
3. Coma or comatose asphyxia.
4. Engorgement of lungs.
5. Numerous bloody points on cut surface of brain.
6. Fluidity of blood.
7. In the chest the lungs are much congested.
8. Heart filled with a large quantity of dark-coloured blood.
9. Great congestion of the sinuses.

⁹⁵ Note

Aparances 1, 3, 4, 5, 6, 7, 8, 9 – Taylor, Alfred, Swaine, *On Poisons in Relation to Medical Jurisprudence and Medicine*, 2nd edn, John Churchill, New Burlington Street, 1859, pp596-597.

Appearance 2 – Christison, Robert, *A Treatise on Poisons in Relation to Medical Jurisprudence, Physiology & the Practice of Physic*, Adam Black & Longman, Rees, Orme Brown & Green, London, 1829, p542.

Chapter 6

Strychnine, A Beneficial Nostrum Or Bitter Bane

6.1 Introduction

Strychnine is normally obtained from dried, ripe seeds of the *nux vomica* plant, which grows in the East Indies. It is also present in the St Ignatius bean, which is a thornless, creeping plant native to the Philippines. Strychnine has been described as the bitterest substance known to man¹ and so, like corrosive acids, would seem to be a difficult substance to use as a poison. Indeed, Sir Robert Christison, the eminent Scottish forensic toxicologist of the nineteenth century, often wondered how anyone could make strychnine the instrument of murder. Despite this, as the cases below indicate, there are means of masking the poison, for example, administering it in foods which have a sour or tart taste or in pills, capsules or cachet.

Thus, despite inherent disadvantages as a poison, cases of deliberate poisoning by strychnine were not rare in the past.² Great problems existed, though, in proving the presence of strychnine by toxicological analysis. Indeed in 1856, in the infamous poisonings committed by William Palmer, a thirty-one-year-old married surgeon of Rugeley in Staffordshire, fierce debate raged over medical attempts to prove that the poisoning of John Parsons Cook had been by administration of strychnine. The physical proof of poisoning was wanting.³ In fact it was plausibly stated, and widely circulated by a portion of the press, before and pending the trial of Palmer, that no man can be held to have died of poison except when poison is found in his body.⁴

The Scottish toxicologist Christison was of the opinion, however, that such a doctrine was erroneous and dangerous. Indeed, he argued that under such a doctrine few criminals would be brought to justice, were they to resort to using strychnine.⁵ Many

¹ See Lonid, N, Banciu, & Bors, G, *Acta Méd Lég Soc (Liège)*, 16, p107.

² Taylor, A.S. *EMJ*, vol 2, 1856-1857, p637, Evidence in Cases of Poisoning.

³ Watson, E.R., *Trial of William Palmer*, Notable British Trials, W Hodge & Co. Ltd, Edinburgh & London, 1952.

⁴ Taylor, A.S., *EMJ*, vol 2, 1856 – 1857, pp636, Evidence in Cases of Poisoning.

⁵ Ibid at p637.

physicians, highly distinguished for their chemical and medico-legal knowledge were not prepared to maintain that the discovery of poison in the body was a sine qua non in every case of alleged poisoning. On the other hand, there were those who believed that as sole evidence of death from poison, chemical tests alone could be trusted. This rationalist optimism aside, the nineteenth century medical profession had severe limitations which contradicted those who desired science to solve all legal questions. Using such chemical tests as existed in the nineteenth century, the accuracy of which could not always be determined, or their value judged, by courts or juries, there was a great risk that a poisoner might not be found guilty.

There existed many reasons for the non-detection of strychnine in the nineteenth and early twentieth centuries.⁶ In 1856 the Edinburgh Medical Journal reported that “strychnine or nux vomica had not yet been found in the tissues of persons poisoned with it.” Further, that “no toxicologist had recorded an instance in which he or others had succeeded in detecting it.”⁷ Moreover, symptoms of strychnine poisoning often resembled tetanus. Therefore, as only very small quantities of strychnine can destroy life, its criminal administration was relatively easy when toxological investigation had not been refined.

Therefore, of the six cases listed below for poisoning with strychnine between 1863 and 1913 making up 6% of total cases, it is of little surprise that a guilty verdict was

⁶ The principal reasons given are – 1. The quantity taken: If the dose be small, from one half to three quarters of a grain, it may be rapidly absorbed and removed from the stomach. It is only the surplus of a fatal dose which is found in the stomach after death. 2. On the time which has elapsed after taking the strychnine, until the symptoms commence: The longer the interval, the greater the quantity of poison removed from the stomach by absorption. The poison has found to be diffused through the circulation in nine minutes. 3. On the careful preservation of the stomach and its contents: If the fluid or solid remaining in the stomach at the time of death is not carefully preserved, there is a great probability, if the residuary quantity be small, that it will not be found. See Taylor, A.S., *EMJ*, vol 2, 1856 – 1857, p644, Evidence in Cases of Poisoning.

⁷ Cook, W.S., *EMJ*, vol 2, 1856 – 1857, p646, The Sale of Poisons.

reached in only one of these cases.⁸ I would submit that most of the verdicts in these strychnine cases were, due to the lack of scientific evidence presented at trial, incorrect.⁹ In England the total number of strychnine cases brought to trial between 1750 and 1914 was forty-one giving a figure of 11% of all criminal cases during that time period.¹⁰ The Old Bailey saw three cases, 6% of all cases, in the time period 1739-1878.¹¹ The inability to detect strychnine arguably contributes to the limited number of criminal trials recorded for the use of this poison. Indeed, a skilful poisoner, acquainted with the selection of poisons, their doses and properties, and having knowledge of compounding them, would often escape the detection of his/her crime entirely in the case of the use of strychnine. Secret murder by strychnine was doubly simple in the past, due to the fact that the poison was easily obtainable by the public. Indeed, until the Pharmacy and Poisons Act of 1933 there existed no restrictions on the sale of *nux vomica* and strychnine.¹² Notable, also, is the fact that under special rules strychnine can still be purchased today as a medicinal ingredient, for research, for chemical analysis, for scientific education or for the killing of moles and seals.¹³ In particular, *nux vomica* is extensively used in homeopathic remedies.

⁸ It should be noted that two of the cases came to trials on the reduced charge of culpable homicide. See Advocate H.M. v Armitage (1885) 5 Couper 675 and Advocate H.M. v Wood (1903) 4 Adam 151. Also in one case where a medical attendant mistakenly prescribed a mixture of equal parts of liquor arsenicalis and liquor strychniae as a remedy for phthisis (pulmonary tuberculosis) to a 36 year old lady who then died from strychnine poisoning no judicial action was taken, as it was difficult to decide whether the doctor or druggist who dispensed the prescription was more to blame - Littlejohn, H.D., *EMJ*, vol XXII, p112, 1907, Three Cases of Fatal Strychnine Poisoning.

⁹ See Conclusion.

¹⁰ Watson, K., *Poisoned Lives: English Poisoners and their Victims*, Hamledon and London, London, 2004, p33.

¹¹ Forbes, T.R., *Surgeons at the Old Bailey*, Yale University Press, London, 1985, Table 8.

¹² All *nux vomica* preparations, except those that contain less than 0.2% strychnine, are subject to the Poisons Rules of 1935, which apply to Part 1, Schedule 1 poisons of the 1933 Act. Strychnine is subject to even more severe restrictions requiring special authority for purchase as set out in Schedule 13 of the 1935 Poisons Rules.

¹³ Rule 17 in Schedule 13 of The Poisons Rules 1935.

Thus, though control of strychnine has increased, it is still a potential weapon for murder. It is also remains the cause of accidental death and suicide.¹⁴

Although the seeds of *nux vomica* had long been known, strychnine itself was not discovered until 1818.¹⁵ By the mid 1800s, potential uses for strychnine were being widely advocated. Beneficent properties were (mostly mistakenly) ascribed to this drug and it was not only widely prescribed, but also its use promoted in many eminent medical journals.¹⁶ Thus, although the chemical and physiological properties of strychnine remained obscure in nineteenth and early twentieth century Britain, it was still widely used in medicine.¹⁷ Strychnine was also readily available for ordinary household use in the form of rat and mouse poisons.¹⁸ This highly toxic poison was, therefore, legitimately on open sale in hardware shops, pharmacies and grocers.

Strychnine stimulates the higher nervous centre leaving the victim fully conscious throughout the experience of poisoning. It is thus an exceptionally cruel poison to inflict on a victim. Initial symptoms include agitation, trembling limbs, stiffness and complete immobility, which is soon, followed by violent convulsions during which the head is bent back, the spine stiffened, the limbs extended and rigid, and the thorax quite immovable. The average fatal period for strychnine poisoning is two hours.¹⁹ In

¹⁴ Between January 1997 and the end of September 2000 there were 19 calls to the Scottish Poisons Information bureau in Edinburgh regarding accidental poisoning with strychnine. Database, Scottish Poisons Information Bureau, Royal Infirmary, Laurieston Place, Edinburgh.

¹⁵ Strychnine is the principal alkaloid in *nux vomica* seeds.

¹⁶ Its use was promoted as a therapeutic agent in various paralytic and nervous affections – Mart, G.R., *EMJ*, vol 47, 1837, pp451 – 452, Practical Observations on the Nature and Treatment of Nervous Diseases, with Remarks on the Efficacy of Strychnine in the Most Obstinate Cases; - Thompson, J., *EMJ*, vol XIX, 1873, Part 1, pp461 – 462, Use and Abuse of Nux Vomica.

¹⁷ For example: Easton's syrup, carthartic tablets, tablets ferrous phosphate. A typical prescription dispensed in 1934 included -: Ammonium carbonate, Tincture of *nux vomica*, Tincture of digitalis, Glycerin and Aqueous acid – One teaspoonful in water twice daily for a Mrs Hastill, 53 East London Street, Edinburgh. This prescription came from one of the bound Prescription Books inherited from my great-grandfather who was an Edinburgh chemist and had a shop in Broughton Street.

¹⁸ Battle's Vermin Killer; Butler's Vermin Killer; Gibson's Vermin Killer; Miller's Rat Powder; Marsden's Vermin and Insect Killer; Barber's Magic Vermin Killer Powders.

¹⁹Tennyson, J.F., *Guy's Hospital Reports*, 3rd series, 1857, p483, On Poisoning by Strychnia.

spite of these tortuous and dramatic effects, by analysis of the six strychnine cases I demonstrate the difficulty of proving instances of strychnine poisoning.

6.2 Aspects of Criminal Negligence

The first case occurred at Perth on the 17th September 1863, when Samuel Tumbleson was tried for attempting to poison his wife Helen, with strychnine. It was alleged that Tumbleson had mixed strychnine with oatmeal and given it to a third party, intending and expecting that his wife would eat the prepared food. The poisoned oatmeal was not, however, eaten by Helen, but fed to a pig which died within a short period of time.²⁰ Medical evidence was led at trial by Dr MacLagan, of Edinburgh, who showed that the oatmeal contained strychnine; this was corroborated by a certain Dr Murray.²¹ Further, a statement from a grocer, Edward Thomson, that Tumbleson had purchased Battle's Vermin Killer in April of the same year proved possession of the poison. Indeed, the poor state of relations between Tumbleson and his wife, along with evidence from various witnesses that he had often wished her ill, appeared to furnish conclusive evidence that Tumbleson was guilty of the crime of which he was accused.²² During the trial, the defence argued that the charge of attempt to murder by means of poison was not relevant on the grounds that the person for whom it was intended had not taken the poison. Despite, however, the presiding judges ruling that this objection was ill founded, the jury returned a verdict of 'Not Proven'.²³

There can be no doubt that the actual finding of strychnine in the oatmeal was a most important link in the chain of proof in this case, as was the fact that Tumbleson had purchased strychnine and wished his wife ill. All principal circumstances for establishing guilt were therefore present, even though the attempt at administration was botched.²⁴ By its very nature, murder by poison is usually a crime done both in secret and with malicious intention. However, it is not in every case that malicious intention is directed against a particular individual. Indeed, this is the situation in the

²⁰ Record of the Lord Advocate AD14/63/270.

²¹ Appendix 21, tests -: A1, A4, A6, A8.

²² Record of the Lord Advocate: AD14/63/270.

²³ HMA v Tumbleson (1863) 4 Irvine 427

²⁴ Possession of poison by accused, secret administration, motive.

next two cases, where the libels were sustained as relevant for culpable homicide. These two cases demonstrate how, despite development in poisons legislation, the public were still not properly protected, and further, they assist in helping to paint a picture of how the law developed in relation to poisons.

By 1868 the Pharmacy and Poisons Act was in force. This Act confined the sale of certain poisons to qualified pharmacists, doctors and druggists who had become members of the Pharmaceutical Society of Great Britain by passing examinations; sale of these poisons had to be recorded in a Poisons Book.²⁵ In more than one respect, however, the Act was incomplete. Any druggist who had been in business before 1868 could be registered with the society upon proof of satisfactory qualifications, and anything not listed as a poison in the Act such as atropine, coca products or carbolic acid could continue to be sold indiscriminately. Further, patent medicines containing mercury, strychnine and morphine required no entry to be made in a Poisons Book. This meant that through carelessness, as in the Buchan case in the preceding chapter, a chemist could make serious errors which could lead to fatalities.

On the 22nd of August 1885, George McLean requested chemist George Armitage of Hamilton Street, Greenock, to supply him with a penny worth of liquorice powder for his mother.²⁶ In culpable violation and neglect of his duty as a chemist, George Armitage dispensed *nux vomica*. Mrs McLean mixed the powder with water drank some of it and in consequence died. During the trial of George Armitage on October 28th 1885 at Glasgow High Court, there was no dispute as to the fact that the deceased had died of strychnine poisoning.²⁷ The question before the jury was whether the

²⁵ The 1868 Act contained two lists- Parts One and Two and included – Part 1- Arsenic and its Preparations, Prussic Acid, Cyanides of Potassium and all metallic Cyanides, Strychnine and all poisonous vegetable Alkaloids and their salts, Aconite and its preparations, Emetic Tartar, Corrosive Sublimate, Cantharides, Savin and its Oil, Ergot of Rye and its Preparations; Part 2- Oxalic Acid, Chloroform, Belladonna and its Preparations, Essential Oil of Almonds unless deprived of its Prussic Acid, Opium and all Preparation of Opium or of Poppies.

²⁶ Liquorice was commonly used in medicine for bronchial problems, coughs, hoarseness, mucous congestion etc. It is often still used in herbal medicine today, but it should be noted that chronic use can lead to hypokalemia (a condition in which the concentration of vitamin K in the blood is low.)

²⁷ HMA v Armitage (1885) 5 Couper 675.

panel was criminally liable for the consequences of his act of dispensing nux vomica instead of liquorice powder.

After a short period of deliberation the jury returned a verdict of ‘Not Guilty’. The unanimous opinion was that there had been an excusable mistake. That a person could escape liability from punishment, because he was able to say he meant no harm demonstrates the distinction in Scottish law between criminal and delictual liability.²⁸ Indeed, the 1868 Act is distinct in stating that it is the duty of a druggist to exercise due care and caution in the dispensing of drugs and medicines.²⁹ In giving consideration to this case, and the fact that smell alone should have distinguished the liquorice from the nux vomica, it is difficult to understand why the jury did not consider the carelessness or recklessness of the act as constitutive of any criminal liability. Given that murder consisted of ‘wicked recklessness’ under Scottish law³⁰, it was impossible to try the druggist on this count. As Farmer writes: “(T)he act of homicide can only be punished according to the level of intention or foresight on the part of the accused.”³¹ Thus, rather than murder, a verdict of culpable homicide would not have seemed unlikely for such a breach of duty of care. It would seem, therefore, that legal enactments, such as the 1868 Act, failed to provide adequate protection for the populace.

The next case in the sequence of this chapter occurred in 1902 when, shortly after nine o’clock on the morning of 13th December, Charles Robson called at the shop of a Mr Anderson, chemist and druggist, in Trinity Road, Leith. Robson handed over a prescription to obtain powders for the treatment of his wife’s neuralgia.³² By half past ten that morning, however, Mrs Edith Robson was dead, having consumed a powder and within five minutes developed symptoms of strychnine poisoning. Post-mortem examination was authorised and carried out by Drs Harvey Littlejohn and T.W.

²⁸ See the English cases of Regina v Noakes [1886] 4 Foster & Finlavson 920; Regina v Spencer [1867] 10 Cox’s Criminal Cases 525 and Scottish case of HMA v Armitage (1885) 5 Couper 675.

²⁹ 32 & 33 Vict. Ch.117, 1868, Poison & Pharmacy Act.

³⁰ See Farmer, L., *Criminal Law, Tradition, and Legal Order: Crime and the Genius of Scots Law 1747 to the Present*, Cambridge University Press, 1996, p143-4.

³¹ Farmer, L., *Criminal Law, Tradition, and Legal Order: Crime and the Genius of Scots Law 1747 to the Present*, Cambridge University Press, 1996, p146.

³² HMA v Wood (1903) 4 Adam 151.

Drinkwater, twenty-six hours after death. From examination both doctors were of opinion that death had been due to strychnine poisoning.³³ In addition, chemical analysis of the stomach and blood was carried out and able to provide evidence of the presence of strychnine.³⁴ The remaining powders were also subjected to chemical testing and each found to contain strychnine.³⁵

As a result of inquiry, it was discovered that an unqualified druggist's assistant, Alexander Wood, had made up the powders.³⁶ Bottles of exalgine and strychnine were found to be stored together in the shop and Wood had dispensed strychnine instead of exalgine.³⁷ Accordingly, Wood was tried on a charge of culpable homicide at Edinburgh High Court on March 17, 1903. During the trial the fact that Mrs Robson had died from strychnine poisoning as a result of the powders was not in dispute. The question for the jury only whether, as in the case of Armitage, the incident been an excusable mistake or culpable negligence.

Evidence was led at trial to show that there had been nothing marked by Anderson to indicate that the bottle of strychnia crystals was poisonous.³⁸ Naturally, this neglectful practice was in contravention of the regulations for dispensing drugs issued by the Pharmaceutical Society.³⁹ The strychnine bottle had, however, the words "Strychnine Crystals" distinctly written on a label attached to the bottle. Since it was a well-recognised rule that there was to be careful scrutiny of all labels on bottles used in dispensing, no error should thus have been made. Indeed, it was common practice in

³³ Appendix 22, appearances:- 5, 6, 7, 10, 14.

³⁴ Appendix 21, tests:- A3, A4, A7, A8.

³⁵ Appendix 21, tests:- A2, A3, A4, A7, A8.

³⁶ Each powder was meant to have been composed of 5 grains of quinine sulphate, 5 grains of phenacetin (a medicine to prevent fever) and 1 grain of exalgine.

³⁷ Exalgine was used for analgesic and anti-neuralgic purposes. It was not a poison under the Pharmacy Acts, and whilst a large dose might be harmful a small dose was not. It was, therefore, not required to be kept in a separate compartment like strychnine.

³⁸ The bottle was not secured on the top and had no distinctive mark, such as skull and crossbones, indicating it contained poison. It was also not kept in a separate cupboard.

³⁹ See also 33 & 34 Vict. Ch. 117, 1868, Poisons and Pharmacy Act.

the past to read a label before and after weighing out “medicines”, and then to spell out every letter on the label to make certain once again.⁴⁰

The omission of the above practice in this case seems extraordinary in circumstances where so many poisons were kept in chemist and druggists shops. In addition, the word “strychnia”, is surely distinctive in itself.⁴¹ Thus, given Wood had eight years experience as an assistant chemist, he had, without doubt, shown gross carelessness. Defence for the panel argued that as the prescription had been dispensed on a dark morning and the strychnine bottle had no distinguishing marks Wood could not be held responsible. Indeed, defence argued that the incident had been merely a regrettable mistake. After a short period of deliberation, the jury concluded that there had been no fault on the part of Alexander Wood – that the panel was ‘Not Guilty’.⁴²

Just as in the Armitage case, upon the evidence, it would seem a surprising verdict and one that denies the fault of the accused in negligently substituting strychnia for exalgine. If Wood had read the label even once it is inconceivable that he would have used strychnine in making up the powders. The court followed Armitage, which implicitly judged that proof of a criminal mind was required for culpable homicide.⁴³ Of great importance in attempting to criticise these verdicts is the fact that in 1899 the Pharmaceutical Society issued regulations, in conformity with the 1868 Act, for the keeping, dispensing and selling of poisons.⁴⁴ Poisons were required to be stored in containers easily distinguishable from those containing ordinary articles. Indeed, the turn of the twentieth century witnessed the development of numerous patent medicine

⁴⁰ Common practice was, therefore, to look at a label three times even in the handling of non-poisonous drugs.

⁴¹ It should be noted, however, that strychnine and exalgine crystallise in the same form and would be very similar in appearance.

⁴² HMA v Wood (1903) 4 Adam 151.

⁴³ HMA v Armitage (1885) 5 Couper 675.

⁴⁴ Pharmaceutical Society’s Regulations for Keeping Poisons 1899 -: Each container of poison had to be labelled with the name of the poison and a distinctive mark indicating it contained a poison. Also each poison had to be kept in a container secured differently from those containing ordinary articles; or in a bottle or vessel rendered distinguishable by touch from other containers or in a container kept in a room or a cupboard separate from ordinary articles.

bottles specifically designed⁴⁵ to prevent errors in dispensing and to protect the populace from accidentally taking poison.⁴⁶ The patent books for these years are overflowing with weird and wonderful ideas to prevent errors in dispensing. Given the verdicts in the last two cases and the obvious non-adherence to Regulations laid down by the Pharmaceutical Society, historical consequences must be considered. Due to the failure of the courts to uphold the importance of these regulations, they failed to protect the Scottish people and failed to enforce due care upon the practice of the sale of drugs. It is highly probable that many others died as a result of the negligence of their chemist.

6.3 Unsatisfactory Verdicts: Judgements of Insanity

A remarkable case, in which the question of guilt remained undecided, occurred in 1906. This is also a case of great historical importance in Scotland, setting a legal precedent which caused much controversy at the time.⁴⁷ On the 19th November 1906, a parcel arrived by post addressed to Mr William Lennox of Old Cumnock, Ayrshire. On being opened, a tin box containing iced shortbread and a card bearing the message “with happy greetings from an old friend” was found.⁴⁸ Mr Lennox, a neighbour, the housekeeper and a servant girl all sampled the shortbread on the evening of November 23rd. Immediately, all were seized with symptoms of strychnine poisoning, with Miss M’Kerrow, the housekeeper, suffering more violently than the others.⁴⁹ Despite attempts by a certain Dr Robertson to assist the housekeeper, she died about an hour and a half after eating the shortbread. The other victims survived.

⁴⁵ For example in 1894 J H Valentine patented a bottle which was either square or rectangular in section. Two of the four sides were raised with pyramid like projections on them and on one side the word POISON was “blown” and on the other there was a representation of a skull and crossbones.

⁴⁶ Some inventors experimented with luminous paints, bells attached to bottles, poison guards, patent poison stoppers and India rubber strips covered in projections. Others created fancy, ornate and incredibly shaped bottles, whose very shape or surface would give a warning message. See Sheridan, M., *Bottles & Bygones*, autumn 1994, vol 1, no.12, p12, Patent Poison Bottles.

⁴⁷ HMA v Brown (1907) 5 Adam 312.

⁴⁸ Record of the Lord Advocate AD15/07/8.

⁴⁹ In general the symptoms included violent twitching and stiffening of the arms and legs.

Post-mortem examination carried out by a Dr McQueen on the body of Miss M'Kerrow provided evidence indicative of strychnine poisoning.⁵⁰ In addition, chemical analysis was carried out by Dr Harvey Littlejohn on the shortbread and viscera and ratified by Dr Murray.⁵¹ This analysis showed the shortbread icing to consist of sugar and 41% strychnine.⁵² Indeed, it was estimated that the total quantity of strychnine present in the icing was not less than 120 grams.⁵³ Analysis of the viscera also showed the presence of strychnine.⁵⁴ Following inquiries, Thomas Mathieson Brown, whose wife was a niece of Mr Lennox, was arrested for the crime on November 28th.⁵⁵

A question of considerable interest arose in connection with the procedure adopted by the Crown in this case. The defence of insanity in bar of trial is familiar enough, but here there was no such special plea in defence, and the insanity of the panel was alleged instead by the Public Prosecutor.⁵⁶ Indeed, the defence maintained that Brown was perfectly sane and should “thole his assize”.⁵⁷

At the first trial diet the Crown accordingly produced a medical certificate signed by two doctors, certifying Brown to be insane and so incapable of pleading.⁵⁸ In respect of this, the case was referred to the High Court for disposal.⁵⁹ During the second diet,

⁵⁰ Appendix 22, appearances:- 1, 4, 5, 7, 13.

⁵¹ As only very small pieces of the shortbread had been eaten, the shortbread cake was practically intact on being sent for analysis. The reason for this was that it tasted so bitter, that it was thought to be bad and put aside.

⁵² Appendix 21, tests:- A2, A3, A7, A8, A10.

⁵³ This large amount explains how such a very small quantity of the cake produced a fatal result in one instance and serious symptoms of poisoning in the others. Half a grain (32mg) can be a fatal dose for an adult, and it is possible that Miss M'Kerrow may have swallowed a grain or more. Indeed, in the shortbread icing there was enough strychnine to have killed several people.

⁵⁴ Appendix 21, tests:- A2, A3, A7, A8, A10.

⁵⁵ Brown was a retired colliery manager of Ardnith House, New Cumnock, Ayrshire. No details, however, are given in relation to any inquiries and of how suspicion fell on Brown.

⁵⁶ Brown had been subject to epileptic fits for forty years and following his arrest the Crown asked for an inquiry to be made by two lunacy experts, into his medical condition.

⁵⁷ Stand his trial.

⁵⁸ This was before the sheriff substitute at Ayr. See also 19 & 20 Vict, Ch.71, S87, 1857, Lunacy Act.

⁵⁹ The panel was, therefore, not called on to plead at the first diet.

at Edinburgh High Court on March 18th -20th 1907 before five Judges, the Crown again argued that the prisoner should not be allowed to plead. Counsel for the defence strenuously opposed this. The legal issue was whether it was a question for the jury to decide if Brown was insane.

The Court held that it was competent for an investigation into the sanity of the panel to precede trial on indictment by jury, and that in the special circumstances of the case it was expedient that the panel should be called on to plead to the indictment. In addition, the Court held that the first question the jury had to determine, after hearing the evidence, was whether the panel could be judged to be sane at point of trial. Should the jury answer that question in the negative, they could not proceed further to determine any question on the merits of the indictment. Brown pleaded ‘Not Guilty’ and evidence was led.

During the trial various facts came to light which clearly pointed to Brown being guilty. The first of these was the purchase by Brown of an ounce of strychnine on May 2nd 1906.⁶⁰ Brown had purchased the poison from John William Sutherland, of the firm of Frazer and Green, chemists, Buchanan Street, Glasgow,⁶¹ claiming that he wanted the poison to kill rats. Thus, the prisoner was proved to have had in his possession poison.⁶²

Further to this, various witnesses deposed that Brown had travelled from New Cumnock to Glasgow, early on the morning of 19th November. Evidence was led to show that whilst there he had purchased un-iced shortbread cake from William Skinner & Sons, Bakers & Confectioners, Glasgow. On his return journey Brown had to change trains at Kilmarnock and was seen entering the post office there. In addition, the brown paper wrappings of the shortbread parcel were identified, at trial, by a post office clerk, Thomas Hart, as being those of a parcel he had handled between

⁶⁰ An ounce contained 437.5 grains. One grain was supposed to be a deadly dose.

⁶¹ An assistant from the shop, Frederick Craig, corroborated the statement of John William Sutherland, as to the sale of strychnine. In addition, an extract was shown from the poisons registry, bearing that one ounce of strychnine was sold to “T.M. Brown Esq” on 2nd May 1906.

⁶² The first principal circumstance for establishing guilt.

11.42am and 11.58am on that day.⁶³ Evidence was then given by two hand writing experts to the effect that both the handwriting of the address upon the parcel and the message card were in the panel's handwriting.⁶⁴

On the day after Brown's arrest, a search of his home produced a small mortar and pestle found lying beside a quantity of icing sugar. Thus, Brown had every opportunity for secret administration of poison.⁶⁵ Further, motive existed in the fact that Brown's wife, being the favourite niece of Mr Lennox, stood to inherit a considerable sum on his death.

The chain of evidence in this case points unmistakably and unerringly to Brown being the man who had dispatched the box which produced such fatal results. Assuming the jury considered the case as proved, they then had to consider Brown's mental state. Throughout the trial evidence was led by countless medical experts. This was in an attempt to prove that Brown suffered from chronic epileptic insanity and was, therefore, of unsound mind.⁶⁶ Given that the court itself stated that "one of the most common influences contributing towards homicidal impulses was the fury that occurred in connection with epileptic attacks", it is of little surprise that the jury returned a majority verdict that Brown was insane.⁶⁷

During this trial much time was given to the vexed question of the accused's insanity. The addresses of counsel and the charge of the learned judge appear inadequate in their respective treatment of whether or not guilt could be inferred. Indeed, the Lord Justice-General practically withdrew this aspect of the case from the jury. Until this case, general practice in Scotland was that the question of a prisoner's sanity was a question based on medical opinion, and decided by a judge or judges. The practice in this case of putting the question of insanity to a jury along with the question of guilt

⁶³ This was due to a certain way of stamping parcels which indicated both time and clerk – HMA v Brown (1907) 5 Adam 312, at p337.

⁶⁴ HMA v Brown (1907) 5 Adam 312, at p335.

⁶⁵ Satisfying the second principal circumstance on which reliance can be placed for establishing guilt or innocence.

⁶⁶ This form of epilepsy was meant to rob the patient of the full control of his faculties, but leave him free to act for a purpose not controlled by a rational and sane will.

⁶⁷ HMA v Brown (1907) 5 Adam 312, at pp338 – 339.

appears irrational.⁶⁸ The jury in this case appears to have had to consider testimony as to the paroxysms of mania and paranoid delusions which suggested Brown's inability to resist or control a morbid criminal impulse. The way, in which questions were formed during the trial to elicit the elements of medical testimony, it is of little surprise that the jury found Brown insane.⁶⁹

Brown was ordered to be detained at His Majesty's pleasure within a lunatic asylum.⁷⁰ Aside from any questions of the accused's insanity, it is worthwhile to weigh up the evidence in this case.⁷¹ That the accused was the donor of the "friendly" gift of poisoned shortbread, there seems no doubt. It is my opinion, therefore, that, *ceteris paribus*, Brown was guilty of the crime of murder by poison. Brown was in many ways perfectly rational, in a condition to speak intelligently on many subjects, and to tell counsel what he did on certain days. Whilst most people inflicted with epilepsy today are easily treatable, many epileptics in the past became asylum inmates. That Brown was considered insane due to his epilepsy certainly must be considered a reflection on medical opinion of the late nineteenth and early twentieth centuries. Indeed, the boundaries between sanity and insanity seem to have been very ill defined.

⁶⁸ The jury was ordered to answer the questions in that order, and on no account move to the second if they found the prisoner insane. This case formed a legal precedent, in that, it was held by a Full Bench that where a question is raised as to whether a person charged on indictment is mentally capable of pleading, the court, both at common law and under the Lunacy (Scotland) Act 1857, had power either – (a) to hold a preliminary inquiry as to the mental condition of the accused before calling on him to plead, or (b) to call on the accused to plead, leaving it to the jury at the trial to say whether he was capable of pleading. Today, although competent to have the question of the accused's fitness to plead decided by a jury, the matter would almost certainly be resolved by some form of enquiry at a preliminary diet.

⁶⁹ As early as 1832, it was claimed that medical evidence materially influences a jury – "If of no value, why are so many witnesses examined?" – Winslow, F., *Westminster Medical School Journal*, 1832, The Principles of Phrenology Applied to the Elucidation and Care of Insanity.

⁷⁰ After the trial Brown was sent to the Criminal Lunatic Department at Perth. In May of that year he was transferred to the Crichton Royal Institute, Dumfries. At the time of his death he was an inmate of Ayr County Asylum.

⁷¹ That the writing of the address on the label of the parcel had an "unfortunate resemblance" to that of the accused was even admitted by Brown's own counsel. That Brown was in possession of the poison employed was also proved. Also that the shortbread was bought in the Argyle Street shop early on the morning on which Brown went to Glasgow, and that he had time and opportunity to post it at Kilmarnock whilst waiting for the local train are all not disputed.

Given that Brown could have been both perfectly sane and also found guilty by a conventional trial, the Crown procedure in this case seems to have been illogical.⁷² All three principal circumstances on which reliance can be placed, as establishing guilt or innocence were, however, present in Brown's case.⁷³ The case was a landmark in Scotland's legal history. Where a question is raised as to the mental capacity of a person charged, Brown set a precedent that a jury could be left to determine whether an accused was capable of pleading.⁷⁴

6.4 Further Unsatisfactory Verdicts

A further accidental case of strychnine poisoning, akin to those mentioned earlier in this chapter, occurred in 1907, when a thirty-six year old lady suffering from a serious heart problem was prescribed an equal mixture of strychnine and arsenic by her doctor to be taken in water three times a day. The lady died due to the contents of this prescription and although both the doctor and the druggist were at fault for the

⁷² Critics have expressed dissatisfaction with the verdict in this case, in as much as the accused having been allowed to plead to the charge, they held that a verdict as to his guilt or innocence should have been returned. If found innocent, Brown would thus have been relieved from the stigma of having committed the alleged crime. See in particular Gane, C & Stodart, C., *A Casebook on Scottish Criminal Law*, W. Green & Son Ltd, Edinburgh, 1988, p304 & Gordon, G.H., *The Criminal Law of Scotland*, W. Green & Son Ltd, Edinburgh, 1967, p331.

⁷³ Possession of poison, opportunity for secret administration and motive.

⁷⁴ "Twill be recorded for a precedent; And many an error, by the same example, will rush into the State." - Shakespeare, *The Merchant of Venice* Act 1V, Scene 1. The current law is, that: Persons who are found insane and unfit to plead are dealt with in the same way as persons acquitted on the ground of insanity. In strict law they are liable to be tried should they regain sanity, but this happens so rarely as to be quite exceptional, although it happened in HMA v Bickerstaff 1926 J.C. 65. One difficulty about the current practice is that it assumes in effect that the person who is unfit to plead committed the act charged, so that he is deprived of any way of establishing his innocence. Even where the plea goes to a jury they are asked to decide on three things- the accused's fitness to plead, whether he committed the act charged, whether he was insane at the time- but if they decide the first in the negative they do not proceed to deal with the other two"- Gordon, G.E., *The Criminal Law of Scotland*, W Green, Edinburgh, 1967, p331. Of interest, is that, according to J.R. Christie, if Brown had been found fit to plead the jury would have acquitted him – Insanity and Recent Criminal Practice (1907) 19 J.R. 165, p171.

prescribing and dispensing of an incompatible and dangerous mixture, no criminal proceedings were taken.⁷⁵

The sixth case in this sequence concerns the trial of John Saunders, a gamekeeper of Gosford, East Lothian, On Wednesday, 23rd April 1913, in the High Court of Justiciary at Edinburgh. The charge was attempted murder of his wife by poison.⁷⁶ Once again this trial failed to produce a ‘Guilty’ verdict. The first witness called at the trial was the alleged victim, Mrs Saunders. Examined by the Solicitor-General, she told of how in January and February of 1913 she frequently became ill after consuming toast with marmalade and at other times wheaten biscuits with cream for breakfast.⁷⁷ In addition, the witness related how the food not only had a bitter taste, but upon it she had often seen “a white powder that glittered in the gaslight.” Both her niece and nurse corroborated this testimony.⁷⁸

The two doctors attending Mrs Saunders also saw and tasted the food and, becoming suspicious, they passed marmalade, cream and biscuits to the police for investigation.⁷⁹ During the trial Professor Harvey Littlejohn read his report of his analysis of the food. All items had shown the presence of strychnine, beyond doubt.⁸⁰ Indeed, the total quantity of strychnine found was 0.323 of a grain.⁸¹ Dr Drinkwater of Edinburgh gave corroborative evidence as to the results of the chemical analysis and both doctors testified that the symptoms suffered were indicative of strychnine

⁷⁵ See Littlejohn, H., *EMJ*, 1907, vol XX111, pp112-113, Three Cases of Fatal Strychnine Poisoning.

⁷⁶ Record of the Lord Advocate AD15/13/16.

⁷⁷ The symptoms she appeared to suffer were characteristic of strychnine poisoning – feeling giddy, shaking, bitter taste in the mouth, choking in throat, twitching in body, rigidity. It seemed, though, that Mrs Saunders suffered from hypochondria. She complained of many things and frequently called on doctors to attend her, whilst also employing a nurse. The doctors, however, could never find anything wrong with her and frequently thought of her as just being lazy- Record of the Lord Advocate AD15/13/16.

⁷⁸ Record of the Lord Advocate AD15/13/16: Testimony of Mary Douglas Chirnside, Mrs Saundier’s niece and Elizabeth Ellen Cameron, the nurse.

⁷⁹ Dr Gamble and Dr Millar of Tranent.

⁸⁰ Appendix 21, tests:- A1, A5, A6, A7, A10.

⁸¹ This amount did not usually account for a fatal dose, but had in the past proved fatal and could have caused death – Record of the Lord Advocate AD15/13/16.

poisoning. There was no doubt during the trial that strychnine had been put in the food.

The search for the perpetrator of this crime was limited as it could only have been a member of the household. By process of elimination, examining facts and motive, only one person could have wilfully put poison in the food, namely John Saunders. No proof of purchase of strychnine by Saunders existed. However, strychnine was, at that time a poison commonly employed by gamekeepers for the destruction of vermin.⁸² It is reasonable to assume that Saunders had strychnine in his possession, leading to a presumption that the first principal circumstance for establishing guilt had been substantiated.

Further, the accused had every opportunity for administering strychnine secretly, being always present in the kitchen when breakfast was being prepared by the niece, thus meeting the needs of the second principal circumstance for establishing guilt.⁸³ As for motive, during their twelve year marriage relations between husband and wife had deteriorated badly. They slept in separate bedrooms, argued frequently and often Saunders was heard to complain bitterly of his wife's constant ill health. In addition, Mrs Saunders had succeeded, through the death of a relative, to a large sum of money. The defence argued that, as Mrs Saunders was a hypochondriac, Mrs Saunders herself, to elicit sympathy, might have introduced strychnine into the food; further, it was stated that a hysterical woman would take even drastic courses of action to excite sympathy. They also argued that Saunders was of high moral character and popular with local people. Following an interval of forty minutes, the jury returned a unanimous verdict of 'Not Guilty'.⁸⁴

⁸² As early as 1857 proclamations were issued by the sheriffs of the counties in Scotland concerning the use of strychnine by gamekeepers. This was due to the number of casualties, which resulted from the poison being placed within reach of the public. See Alison, A., *EMJ*, vol 2, 1856-1857, p286, Use of Strychnine by Gamekeepers.

⁸³ The accused would be left alone in the kitchen, whilst his wife's niece firstly carried up a breakfast tray to her grandmother, Mrs Saunders mother.

⁸⁴ "The jury, thank heaven! Does not content itself with a moral conviction. The strongest probabilities cannot draw from them an affirmative verdict." – Gabordiau, E., *The Widow Lerouge*, 1873, Osgood, Boston, reprinted by Anno Press, New York, 1976.

6.5 Contemporary Contrasts and Conclusions

The most recent case of murder by strychnine for which information is extant occurred in 1965. It concerned the murder of nine year old William Hendry by Samuel Alexander Coltart in Greenock.⁸⁵ Of course, this case falls outside the selected time period, but is worth mentioning because it highlights the progress of forensic toxicology in cases of strychnine poisoning and the limits imposed by scientific fallibility on nineteenth century trials. Post-mortem examination in this case provided striking evidence, and strychnine was also found in ante-mortem samples.⁸⁶ Coltart was found ‘Guilty’ by a majority verdict and given a life sentence.⁸⁷ This case has never been reported in law reports or legal journals.⁸⁸ It is important, though, as it affirms that poisoning was still a crime that occurred in the twentieth century.⁸⁹

Early writings on the subject of toxicology were largely speculative and theoretical, while the practical use of scientific investigation in crime was only in its infancy in the nineteenth century. It was in as late as 1833 that the chemist, James Marsh, first gave expert evidence in a criminal trial based on the test he had developed for the detection of arsenic.⁹⁰ Medical jurisprudence and toxicology were minor disciplines until the early twentieth century; and medical evidence was not, as we have seen in the chapter thus far, deemed universally reliable in courts. Indeed, the facility and simplicity of operation by which most poisons may now be recognised with certainty stands out starkly from that which could be obtained fifty or one hundred years ago.

⁸⁵ Glasgow Herald, September 9th, 1965, p9 & Glasgow Herald, September 10th 1965.

⁸⁶ Appendix 22, appearances -: 5, 7, 8, 10, 11, 14 & Appendix 21, tests -; B1, B2.

⁸⁷ In the words of Lord Leechman, the presiding judge at the trial “There is only one sentence I can pronounce and that is imprisonment for life.”- Glasgow Herald, 10th September, 1965.

⁸⁸ Fortunately the Crown Office in Edinburgh was able to provide me with a copy of the indictment for this case, from which I was able to find related articles in the Glasgow Herald.

⁸⁹ See for example the case of Margaret Veitch or McMillan who was charged with murdering her husband with arsenic in 1940 at their farm in Kirkintilloch although a ‘Not Proven’ verdict was reached –Glaister, J., *The Power of Poison*, Christophehr Johnson, London, 1954, pp213-235.

⁹⁰ Watson, K., *Poisoned Lives; English Poisoners and their Victims*, Humbledon & London, 2004, p207.

It is of little surprise, therefore, that wrongful verdicts seem to have been reached in so many poisoning trials during the 1800s and early 1900s, and in particular, amongst these six strychnine poisoning cases in the time period 1800 to 1913. Indeed, according to Alfred Swaine Taylor, writing in 1856, colour tests for the detection of strychnine were not only uncertain but it was his belief that chemical analysis was not always necessary to secure conviction on a poisoning charge and that there could be a conviction on medical and circumstantial evidence alone.⁹¹

As regards the appreciation of forensic medicine by the general public, ignorant of the modern progress of science, they - following the Latin principle, “that everything unknown is taken for great” – “were rather astounded by the evidence given in the courts by experts.”⁹² It is, therefore, not surprising that a jury of fifteen, when confronted with chemical results and accurately noted pathological appearances, would err on the side of caution when reaching a verdict in a trial for murder by poison.

Further, in many instances during the nineteenth century it was the evidence of the general practitioner, not that of the special expert, on which the courts depended for the primary information, which was essential for their guidance. In such instances where the ordinary medical practitioner did not regard medical questions in a forensic light, important points of observation would have been missed or mistakes made which may have led to miscarriages of justice. In addition, only a limited number of persons were engaged in medico-legal practice in the past; it was unnecessary to ask a special knowledge of it from everyone who entered the medical profession.⁹³ It is

⁹¹ See Gibert, J., Report of Trial of William Palmer, London, May 14, 1856 per Dr Christison. See also testimony of Dr Taylor when being examined in chief during this trial –

Question – “Are not the colour tests of strychnine so uncertain and fallacious that they cannot be depended upon?” Answer - “Yes, unless you first get the strychnine in a visible and tangible form.” Browne, G.L. & Stewart, C.G., *Reports of Trials for Murder by Poisoning*, Steven & Sons, London, 1883, pp85-231.

⁹² Maclagan, D., The *Journal of Jurisprudence*, vol XXIII, 1879, p2, Forensic Medicine from a Scottish Point of View.

⁹³ Medical Jurisprudence became a compulsory part of medical examinations at Edinburgh University in 1833. In 1839 forensic medicine was introduced as a compulsory part of the medical examinations at Glasgow University.

unlikely, therefore, that the ordinary medical practitioner would have been able to distinguish between idiopathic tetanus and strychnine poisoning.⁹⁴ Also given there are no typical post-mortem appearances in cases of strychnine poisoning, proper chemical analysis was required to provide proof of poisoning by strychnine in a court of law. Whilst medical jurisprudence and toxicology appear to have a long history; this history is peculiar in that it seems to have possessed a literature before it had acquired a really practical use and application. There existed no certainty in the tests provided in the textbooks for strychnine and other poisons such as opium and phosphorous during the nineteenth and early twentieth centuries.

Despite the chemical analysis of the presence of strychnine in the preparations administered (or intended for administration) in the above cases, scientific ability to test for strychnine did not translate into ‘guilty’ verdicts. Given the prevalence of strychnine in society and the reliance on circumstantial facts by juries, medical evidence in the past was just not thought of highly enough to be considered as conclusive proof of an attempt to murder.

⁹⁴ Idiopathic tetanus arose from exposure to cold and wet.

APPENDIX 20

Strychnine Poisoning Cases 1863 -1965

Date	Place	Accused	Victims(s)	Medium of Administration	Excuse for Obtaining Strychnine	What Happened to Victim(s)	Trial verdict
1863	Dundee	Samuel Tumbleson	Wife- Elizabeth Holland	Oatmeal	Killing Rats	Survived	Not Proven
1885	Greenock	George Armitage	Jane McLean	Medicine	Not Known	Died	Not Guilty
1902	Edinburgh	Alexander Wood	Edith Robson	Medicine	Not Known	Died	Not Guilty
1906	Ayrshire	Thomas Mathieson Brown	Grace McKerrow	Shortbread	Killing Rats	Died	Insane- Detained as His Majesty's Pleasure
1907	Edinburgh	Druggist Doctor	Lady-36 (Name Unknown)	Medicine	Not Relevant	Died	No Legal Action Taken
1913	East Lothian	John Saunders	Wife- Elizabeth Saunders	Marmalade, Cream, Biscuits	Not Known	Survived	Not Guilty
1965	Greenock	William Coltart	William Hendry	As Powder	None Theft	Died	Guilty Life Sentence

APPENDIX 21

Historical Tests Employed For Detecting Strychnine

A. Before 1915⁹⁵

1. Colour Test. – Add two drops of strong sulphuric acid to crystals and touch edge of solution with a particle of manganese dioxide or potassium dichromate. If strychnine, a deep blue colour forms at the point of contact which rapidly changes into purple, crimson, rich red-brown, then fades into bright orange-red which remains for some hours.
2. Physiological Test. – Inject the suspect material into the dorsal lymph sac of a frog. Strychnine will cause a convulsion after a few minutes.
3. Neutralise suspect solution with carbonate of potash and evaporate to dryness. Wash residuum with warm rectified spirit to near dryness and then moisten with sulphuric acid. Add small fragments of crystals of bichromate of potash and a deep violet colour will be observed if strychnine present, which passes into red.
4. Taste Test. - Neutralise suspect liquid with carbonate of potash and evaporate to dryness. Treat dry extract with warm rectified spirit and filter. On tasting will perceive bitterness if strychnine present.
5. Taste Test. – The most prominent physical characteristic of strychnine is its bitter taste. One grain of strychnine in a gallon of water is distinctly perceptible.
6. Taste Test. - Dissolve suspect material in water with a trace of acid. Cautiously taste and if bitterness strychnia probable.

⁹⁵ Note

Tests 1, 2, 5 – Glaister, John, *Medical Jurisprudence and Toxicology*, 12th edn, E & S Livingstone Ltd, London, 1962, p695.

Test 8 – Littlejohn, Harvey, Duncan, “Three Cases of Fatal Strychnine Poisoning”, *EMJ*, vol XX111, 1907, p118.

Tests 3, 10, 11 – Scholefield, William, “Case of Poisoning with Strychnia”, *EMJ*, 1868-1869, vol 14, pp410-412.

Test 7 – Sigmond, George, “Lectures on Materia Medica and Therapeutics; Nux Vomica, p864.

Tests 4, 6, 9 are taken from Records of The Lord Advocate.

7. Add small quantity of nitric acid to suspect material. If strychnine present, deep red colour obtained.
8. Bichromate of potash solution gives with strychninia, at once or on standing, a yellow precipitate, appearing under the microscope as rectangular plates and prisms.
9. Sublimate of strychnia touched with a drop of dilute picric acid solution, strength 1 in 250, gives microscopic arborescent crystallizations of peculiar curved forms.
10. Colour Test. - Treated with concentrated sulphuric acid and then with a crystal of sodium nitrite, strychnine gives a dirty yellow colour, changed by an alcoholic solution of potash to a fine orange-red.
11. Colour Test. – Strychnine will give an instant yellow colour when treated with a trace of nitric acid.

B. After 1915⁹⁶

- 1.** Thin layer chromatography
- 2.** Ultra-violet spectrometry

⁹⁶ **Note** Chromatographic analysis was first employed by the chemist Tswett in 1906 to separate the various pigments in leaves. The full possibilities of this method have been appreciated only since about 1935. The ultra-violet spectral region was discovered by the chemist Lyman in 1915.

Note

Tests 1, 2 – Bogan, J.R., Rentoul, E., Smith, H., & Weir, W.P., “Homicidal Poisoning by Strychnine”, *The Forensic Scientist*, vol 6, 1966, pp166-167.

APPENDIX 22⁹⁷

Post Mortem Appearances

1. Fluidity of the blood.
2. Emptiness of the throat.
3. Congestion of the dura-matter, or outer membrane of the brain.
4. Face cyanosed.
5. Lower limbs rigid.
6. Arms stiff and flexed.
7. Body quite stiff and rigid.
8. Pupils dilated.
9. Abdomen distended.
10. Lower jaw firmly oppressed to upper jaw.
11. Fingers semi-bent and hook like.
12. Anatomical lesions of stomach.
13. Engorgement of the lungs.
14. Engorgement of the vessels of the brain and spinal cord.
15. Lower jaw firmly appressed to upper.

⁹⁷ Note

Appearances 1, 2, 3 - Taylor, Alfred, Swaine, "Evidence in Cases of Popisoning", *EMJ*, vol 2, 1856-1857, p644.

Appearances 5, 6, 7, 8, 10, 12, 13 – Littlejohn, Harvey, "Three Cases of Fatal Strychnine Poisoning", *EMJ*, vol XX111, 1907, p114.

Appearances 9, 11, 15 – Scholefield, William, "Case of Poisoning with Strychnia", *EMJ*, 1868-1869, vol 14, p411.

Appearances 4, 14 are taken from Records of The Lord Advocate.

Chapter 7

Prussic Acid, A Popular Paregoric or Pernicious Potion

7.1 Introduction

During the early 1800s, advances in chemistry led to the isolation of many medicinal preparations from simple substances. The isolation of the alkaloids, in particular, was an important event in the development of pharmacology.¹ Among the new remedies of medical practitioners in the 1800s, prussic acid achieved a highly regarded reputation and its use was much promoted in medicine.² It was also used extensively in industry and as a fumigating agent.³

Prussic acid, or hydrogen cyanide, is the principal active ingredient found in bitter almonds (*prunus amygdalus*), the leaves of the cherry-laurel tree (*prunus laurocerasus*), the peach blossom (*prunus persica*) and several other vegetable substances.⁴ The use of prussic acid in medicine is very ancient and accounts of sudden death as a result of poisoning suggest that prussic acid has been available for assassins for many centuries, probably in the form of aromatic preparations of peach

¹ An alkaloid is a chemical substance found in various plants.

² Medical matters for which prussic acid was used included dyspepsia, cholera, gonorrhoea, epilepsy and night coughs in children. See Elliston, J., *Lancet*, vol 2, 1826 - 1827, pp761-762, Hydrocyanic Acid in Dyspepsia; Stuart, J., *EMJ*, vol 28, 1827, pp271-277, On the Use of Hydrocyanic Acid in Chorea; Haynes, R.H., *Lancet*, vol 2, 1828 – 1829, p159, Prussic Acid as a Remedy in Gonnorrhoea; Copland, P., *Lancet*, vol 1, 1832 – 1833, pp346 -349, Nature and Treatment of Epilepsy; MacDonald, K.N., *EMJ*, vol XXIV, 1879, pp981-986, On the Therapeutic value of Hydrocyanic Acid in Arresting the Night Cough of Children after Failure with the Bromides.

³ Prussic acid was used as a reagent in dyestuff manufacture, tanneries, fertilizer plants, gold mining, gilding and photography. It was also used to free rooms from bugs and lice and to fumigate ship's holds to rid them of vermin- Simpson, K., *Taylor's Principles and Practice of Medical Jurisprudence*, 12th edn, Churchill, London, 1965, p373.

⁴ Prussic acid is also known as hydrocyanic acid or hydrogen cyanide. When pure it is a colourless, volatile liquid. It consists of hydrogen 34.5%, carbon 24.8% and nitrogen 40.7% - Ibid, p373.

and almond.⁵ In early *materia medica* there were a number of preparations containing hydrocyanic acid. Amongst these was ‘cherry-laurel water’, prepared by distilling the leaves of the plant. It was an investigation into the toxicity of this preparation that prompted modern pharmacological study of prussic acid⁶

7.2. Prussic Acid As A Poison

In 1731, Thomas Madden a lecturer in anatomy and surgery at Trinity College, Dublin, published the results of an investigation into the effects of cherry-laurel water, which, used as a flavouring additive to brandy, had caused the deaths of two Dublin women. Madden’s experiments were on dogs, and he showed that reaction varied with dose: small doses caused convulsions; larger doses brought about paralysis and rapid death.⁷ Further confirmation of the poisonous nature of cherry-laurel water appeared in medical journals in 1737 as a result of experiments carried out by the chemist Abraham Vater.⁸

The fact that prussic acid is frequently found in the plant kingdom makes it a poison of particular medico-legal interest because, in a poisoning case, the defence may set up the argument that the poison has been ingested in the normal intake of food.⁹

⁵ The oil derived from bitter almonds, peach blossom and the cherry-laurel tree have sedative properties and were used in cough remedies. In addition, the cibated layer of the black cherry was an official preparation in the pharmacopoeias of London and Edinburgh colleges during the early 1700s until the experiments of certain physicians in Worcester demonstrated its fatal effects. See Pemberton, H., *The Dispensary of the Royal College of Physicians*, London, 1746, p77. See also Orfila, M.J.B., *EMJ*, vol 33, 1830, pp220-222, On the Detection, Morbid Appearances, and Treatment of Poisoning with Hydrocyanic Acid.

⁶ Earles, M.P., *History of Medicine Journal*, vol 3, 1966, pp305-313, The Introduction of Hydrocyanic Acid into Medicine.

⁷ Madden, T., *Philosophical Transcripts of the Royal Society*, Royal Society of Chemists, vol 37, 1731, p84.

⁸ Earles, M.P., *History of Medicine Journal*, vol 3, 1966, p306, The Introduction of Hydrocyanic Acid into Medicine.

⁹ See Reg v Tawell [1845] Aylesbury Spring Assizes, reported by Browne, G.L., & Stewart C.G., *Reports of Trials for Murder by Poisoning*, Steven & Sons, London, 1883, pp 16-49.

Indeed, accidental poisoning by fruit kernels was at one time not uncommon.¹⁰ It is noteworthy that it still on occasion occurs today.¹¹

To date there have been only four trials in England and one in Scotland (2% of all cases) for murder with prussic acid.¹² Due to the comparative ease however with which prussic acid could be obtained and administered, and also considering the fact that it is among the most rapid and deadly of poisons, it is likely that many more incidences of deliberate prussic acid poisoning have been undetected. Indeed, until the passing of the Pharmacy and Poisons Act of 1868 there was little control over the sale of prussic acid.¹³ Prussic acid was often not even considered a poison during the early nineteenth century. A writer in *The Lancet* in 1833, after prescribing prussic acid for eye disease and deafness, complained that “the apothecary had the unprofessional audacity to put upon the bottle the word poison.”¹⁴

¹⁰ Ibid, pp 16-40, pp50-60, pp233-270.

¹¹ Between 1997 and 2000 there were six calls to the Scottish Poisons Information Bureau regarding poisoning with fruit kernels- one call each re: peach stone, plum stone and olive kernels. Also three calls re: cherry laurel (the plant). This information was given by personal inquiry. Scottish Poisons Information Bureau, Royal Infirmary, Laurieston Place, Edinburgh, EH3 9YW.

¹² See English trials of Captain John Donellan in 1781 for murder of Thomas Broughton- Forbes, T.R., *Surgeons at the Old Bailey*, Yale University Press, London, 1985, pp155-158; Trial of a certain Mr Freeman in 1829 for the murder of Judith Buswell- Sigmond, G., *Lancet*, vol 2, 1836-1837, pp795-796, Lectures on Materia Medica and Therapeutics; Trial of John Talwell for murder of Sarah Hart in 1845, and trial of George Ball for poisoning his mother in 1860- Brown, G.L., & Stewart C.G., Steven & Sons Ltd, 1883, pp16-52. The Scottish trial is that of John Thomson in 1857 – Record of the Lord Advocate AD14/57/123.

¹³ Even after the passing of this Act prussic acid could still be purchased easily in any chemist’s shop as it was often in for example lotions used to treat chilblains or varicose veins. It was not until the Poisons and Pharmacy Act of 1933 that further restrictions were put in place. Under the 1933 legislation, however, members of the public who satisfied the requirements of the Poisons Rules governing Schedule 1 poisons of the 1933 Act could still obtain Scheele’s acid (prussic acid) for photographic processes, electro-plating or for the destruction of wasps. It is noteworthy that in modern times in Missouri, Colorado, Nevada, North Carolina and California prussic acid is used in the death chambers for executing criminals

¹⁴ Epps, J., *Lancet*, vol 2, 1832-1833, p699, Hydrocyanic Prescriptions: - “I shall now mention a circumstance which shows either the grossest ignorance or the wicked disposition on the part of an apothecary.”

Nevertheless, the symptoms for poisoning with prussic acid include collapsing, convulsions, unconsciousness and sometimes frothing at the mouth. Aiding the concealment of a crime of prussic acid poisoning is the fact that these symptoms could easily have been mistaken in the nineteenth century for epilepsy. Further, chemical tests for prussic acid in the 1800s were both conjectural and imperfect, with much argument in medical journals regarding accuracy.¹⁵ Prussic acid is also extremely volatile and can become untraceable within a very short time period or may easily convert into a different substance during putrefaction of a corpse, meaning that chemical testing had to be done immediately; otherwise negative results would be obtained.

Although prussic acid was more expensive than arsenic or strychnine, and has a distinctive taste and odour of bitter almonds, it would appear that it has often not received the attention bestowed upon other poisons. Perhaps this is due to the fact that the pronounced taste and smell from prussic acid could not in any way be disguised. Of note though is that not every person in the population is able to recognise this distinctive smell and indeed it has been said that only fifty percent of the population are able to detect this odour.¹⁶ Speculation should therefore be given as to how often prussic acid was used as a means of murder, albeit undetected. A lack of trials for murder with prussic acid does not mean that murder with prussic acid did not occur, and it is possible that there were many undetected incidences of poisoning with prussic acid in the past, especially given the difficulty of post-mortem detection or correct diagnosis of symptoms.

7.3 Prussic Acid Poisoning Trials in the Mid-Nineteenth Century

The only Scottish case in the Records of the Lord Advocate is that of John Thomson who was tried on the 22nd December 1857 at the Circuit Court of Justiciary in

¹⁵ In particular see Orfila, M.J.B., *EMJ*, vol 33, 1830, pp221-223, On the Detection, Morbid Appearances, and Treatment of Poisoning with Hydrocyanic acid.

¹⁶ Prussic acid when pure gives off a strong odour of peach-blossom, cherry laurel water or bitter almonds. Not everyone is able to detect this smell. See Polson, C. J., & Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London, 1973, p147. See also Watson, K., *Poisoned Lives; English Poisoners and their Victims*, Hambledon & London, 2004, p29.

Glasgow for both murder and attempted murder with prussic acid. The indictment was unusual in that the panel was charged firstly with the distinct crime of murder, and secondly with attempt to poison.¹⁷ Though this double indictment would appear to be of both historical and legal importance in the history of poisoning in Scotland, at the time however the case appeared to generate little academic interest. Naturally, a certain amount of public saturation with respect to such issues had occurred due to the extensive media interest afforded to the trial of Madeleine Smith five months earlier. It was from newspaper reports of the Madeleine Smith case, though, that Thomson acquired much of his useful knowledge of poisons and was perhaps even inspired.¹⁸

The setting for this crime was the village of Eaglesham, nine miles from Glasgow. Here Thomson, a tailor, occupied a room in a two-storey tenement.¹⁹ Living in a room on the upper floor was Agnes Montgomery, a twenty-seven year old who worked as a reeler in the local cotton mills.²⁰ For some months Thomson had admired Agnes and had often proposed marriage. She, however, always declined. The couple remained

¹⁷ These crimes were charged as committed against the same individual although they did not seem in any way to be related. Usually such acts should “have a natural relation and dependence as parts of one foul and nefarious story.” – Hume, D., 1819, *Commentaries on the Law of Scotland, Respecting Crimes*, vol 2, Bell & Bradfute, Edinburgh, 1819, p166 . Note also that counsel for the panel neither objected to the indictment nor moved for separation of the charges, though had he done so, as appears from an observation from the Justice – Clerk, his Lordship would have sustained the objection and proceeded with the first charge alone- Roughead, W., *Glengarry’s Way and Other Stories*, W Green & Sons Ltd, Edinburgh, 1912, p130.

¹⁸ It has been suggested that newspaper reports of murder were “accessories before the fact to three-fourths of the more extravagant murders that occurred in England.”- “A murder occurs the journalist does his work; and the poison he gives forth floats over the country like a pestilence.” – Altick, R.D., *Victorian Studies in Scarlet*, LM Dent & Sons Ltd, London, 1972, p299.

¹⁹ John Thomson was actually an alias for Peter Walker, a 26 year old native of Tarbert, Argyllshire. In 1853 Peter Walker, namely John Thomson, had been sentenced for stealing £22 from his employer, hence his adopting a new name. Information in relation to this is given in the details of his poisoning case- Record of the Lord Advocate AD14/57/123

²⁰ Agnes was the sister of a Mrs Watson who lived downstairs with her tailor husband, James, and their 3 year old daughter Janet A reeler in the cotton mills was someone who unwound yarn from bobbins and rewound it on to a revolving reel in the form of skeins or hanks – See www.linkforlife.org, *Textile Industry*, p3.

apparently on friendly terms and often Thomson would visit Agnes on a Sunday afternoon.

On Sunday 13th September 1857, Agnes was in her usual good health and spirits.²¹ At about ten to five she was visited in her room by Thomson and young Janet Watson, daughter of Agnes's sister. Then at five o'clock, eight year old Janet McGregor, a neighbour's child, knocked at the door with a message from a friend of Agnes asking if she wanted to go for a walk.²² Agnes replied that she would, but firstly could Janet McGregor fetch a bottle of table beer.²³ This she duly did, noting that Thomson and the child, Janet Watson, were still with Agnes on her return.

At roughly ten past five two neighbours heard a heavy fall in Agnes's room and then saw Thomson and the little girl quickly depart.²⁴ Shortly afterwards, a loud moaning was heard coming from the tenement by a Mrs McDonald and Janet McGregor's sister, both of whom were at the nearby well. They made their way inside and on reaching the landing in the tenement found that "the moans were from Aggie's room."²⁵ On entering the room Mrs Watson, noticed a peculiar smell, like bitter almonds. Describing the scene, she said that "Aggie was sitting on a chair before the clock, her head leaning to the right on the table, her right hand hanging down, the left in her lap. A thick slaver was coming from her mouth and her eyes were staring. She was alone."²⁶

Thomson immediately offered to fetch the local surgeon, Dr Scott, who, on arrival, assumed this to be a case of apoplexy.²⁷ Dr Scott attempted bleeding. This was,

²¹ Agnes had talked with her sister Mrs Watson and a friend, William Young, at the close mouth about half past four that day. According to the precognitions of both of these witnesses she was then quite well- Record of the Lord Advocate AD14/57/123.

²² The friend was a Miss Dollar who lived nearby.

²³ Agnes gave Janet a three penny piece and the child fetched a bottle of beer from Dollars' - the father of her friend. Dollar had a liquor licence.

²⁴ These neighbours were a Mrs Clarkson and a Mrs Law- record of the Lord Advocate AD14/57/123.

²⁵ Record of the Lord Advocate AD14/57/123.

²⁶ Record of the Lord Advocate AD14/57/123.

²⁷ Apoplexy is a term introduced by Hippocrates meaning a stroke - Comrie, J.D., *Black's Medical Dictionary*, 8th edn, A & C Black Ltd, 1926, p60.

however, unsuccessful and so Dr Scott tried to administer a toddy, as a pick me up,²⁸ but could not.²⁹ The doctor then administered a turpentine enema, but by six o'clock Agnes was dead. She was buried on Thursday 17th September.³⁰ The cause of death given on the death certificate, as filled out by Dr Scott, was apoplexy. At no point in time did anyone in the village question the accuracy of this judgement. Indeed, there was no suspicion of foul play.

A week later, on Friday 25th September, Thomson left Eaglesham, and made his way to the home of a respectable couple named Mason, in John Street, Glasgow, with whom he had previously lodged.³¹ On his arrival, just before midnight, Thomson produced a bottle of whisky and offered a drink to his hosts.³² Mr Mason took a little whisky and felt that it had a strange, bitter taste.³³ He handed back the glass and Thomson filled it for Mrs Mason who drank nearly a half of it. Immediately, Mrs Mason complained of a strange taste, began to feel dizzy, began to suffer double vision and lost the power of her limbs.³⁴ During a previous visit Thomson had stolen

²⁸ A toddy is a drink which originated in Scotland and usually consists of whisky, sugar, lemon juice and water. It is often used in the treatment of common colds, where it is served hot. It is also believed to be a useful “pick me up.” In the words of Dr Scott “Her face had a distressed look, her eyes were fixed and staring, she seemed to be breathing with considerable difficulty and there was some frothy mucus streaked with blood about her mouth.” – Record of the Lord Advocate AD14/57/123.

²⁹ This was, because, Agnes's teeth were so tightly clenched together- Record of the Lord Advocate AD14/57/123.

³⁰ The duration of the “illness” had been under fifty minutes. Noteworthy, is that Dr Scott did not send notice to the local police constable of the sudden death. At the trial of Thomson the Lord Justice Clerk recommended Dr Scott to do so in any future cases- Cowan, S., *EMJ*, vol 3, 1857-1858, p937, Eaglesham Poisoning Case.

³¹ A letter containing money had disappeared and Thomson's employer in Eaglesham suspecting him of this act, had dismissed him – Record of the Lord Advocate AD14/57/123.

³² Thomson had left Eaglesham at seven o'clock that evening and walked to Glasgow. During his journey Thomson called at spirit dealers in Clarkston and purchased a pint bottle of whisky. On arrival at the Mason's home Thomson explained that he had come back to occupy his former lodgings and to celebrate had brought the whisky.

³³ Mr Mason felt his face immediately flush and refused to take any more- Record of the Lord Advocate AD14/57/123.

³⁴ Record of the Lord Advocate AD14/57/123. Mrs Mason, in fact, remained ill for several days, but ultimately recovered.

clothes belonging to another lodger and so, the following morning, Mr Mason went to the local police station to inform them of Thomson's return. As a result of this Thomson was taken to the local prison, where he remained until September 29th.³⁵

Meanwhile on September 29th, Janet Watson, who had accompanied Thomson on his visit to Agnes Montgomery's lodgings earlier in the month, related to her mother that "John gave aunty ginger and she fell on the floor and spued". Alarmed by the child's recollection, Mrs Watson became suspicious that the death of Agnes may not have been from apoplexy.³⁶ Therefore, she set out next morning for the court house in Glasgow and there informed a detective officer that she believed Thomson had murdered her sister.³⁷

Thomson was duly arrested and on 1st October brought before the sheriff-substitute and charged with the murder of Agnes. During inquiries into the case the authorities had been alerted to the incident with the whisky at the Mason home and as a result of this Thomson again appeared before the sheriff-substitute on October 21st, charged with attempting to poison Mr and Mrs Mason.³⁸

The body of Agnes Montgomery was exhumed on 30th September, by virtue of a warrant from the sheriff of Renfrewshire. A post-mortem examination was carried out by Drs Daniel and Walter M'Kinlay of Paisley (father and son), assisted by Dr Scott of Eaglesham. From the advanced stage of decomposition of the body the doctors were unable to assign with certainty the cause of death.³⁹ It was evident; however, that death was neither from external violence, protracted disease, nor structural changes in

³⁵ Thomson was released on Tuesday the 29th, there being insufficient evidence and no trace of the stolen property.

³⁶ Record of the Lord Advocate AD14/57/123.

³⁷ By coincidence Thomson was then in court, having a liking for being a spectator during criminal trials. Mrs Watson pointed him out to the detective and he was arrested on the spot.

³⁸ Possible motive for this was that Thomson did not wish to be charged with the theft of clothes during his previous visit.

³⁹ Appendix 25, appearances -; 2, 3, 6, 9, 13, 15, 17, 20, 25, 26.

any of the important organs.⁴⁰ Significant though was the smell of bitter almonds perceived on opening the body.⁴¹ There were no symptoms of death by apoplexy.⁴²

Certain portions of the body were removed for chemical examination by the Drs M'Kinlay. From eleven different tests carried out, both doctors concurred that not only was prussic acid present in the stomach, but that it had been the cause of death.⁴³ Further experiments were carried out by the prominent forensic toxicologist, Dr Maclagan of Edinburgh on the viscera, which confirmed these findings.⁴⁴ To be noted, however, is that whilst the Doctors M'Kinlay found no prussic acid in the spleen, Dr Maclagan had clearly detected the presence of prussic acid there. Also, Dr Maclagan detected a much lesser amount of prussic acid in the stomach than the Doctors M'Kinlay.

During the trial of Thomson at Glasgow High Court in December 1857 further medical evidence was given to show that the black pint bottle of whisky left in the Mason household had contained such a proportion of prussic acid as to constitute one glass of wine of the mixture, a fatal dose.⁴⁵ The intentions of Thomson were thus established beyond doubt.⁴⁶ The defence attempted to make the argument that the prussic acid in the body might simply be the result of processes of decomposition - a

⁴⁰ Cowan, S., *EMJ*, vol 3, 1857-1858, p939, Eaglesham Poisoning Case- Medical Evidence.

⁴¹ See Appendix 25, appearance :- 13.

⁴² "Where death resulted so quickly, there would certainly, if apoplexy was the cause of death, have been effusion of blood, and there was not, in the post-mortem appearance, the slightest trace of any such thing." – Cowan, S., *EMJ*, vol 3, 1857-1858, p939, Eaglesham Poisoning Case- Medical Evidence.

⁴³ Appendix 24, tests :- 1, 2, 3, 4, 5, 6, 10, 12, 14, 15, 16.

⁴⁴ Appendix 24, tests :- 1, 2, 3, 10, 12.

⁴⁵ Appendix 24, tests :- 1, 3, 12, 16, 17. By precipitation with silver nitrate (test 1) it was shown that in each fluid ounce of the whisky was present 0.87 grains of prussic acid.

⁴⁶ Professor Penny a forensic toxicologist in Glasgow who had given testimony for the Crown in the Madeleine Smith case confirmed the evidence of the medical witnesses during the trial and confirmed the appropriateness of the chemical tests employed for detection of prussic acid.

theory put forward by the renowned forensic toxicologist, Orfila.⁴⁷ The court rejected these claims and as it was concluded that this was mere speculation on the part of Orfila.⁴⁸

The trial of John Thomson presented several points of interest. It very speedily followed the still more celebrated trial of Madeleine Smith; it was the first in Scotland where prussic acid had been employed as the poisonous agent; and it formed the third in a short series of British trials for poisoning with prussic acid, where a conviction was obtained.⁴⁹ Discrepancies among the medical witnesses for the prosecution in relation to the detection of prussic acid in the body contributed to the panel's defence.⁵⁰ Despite this, however, the circumstantial evidence against Thomson was very strong. Indeed, even had the poison which deprived Agnes Montgomery of life never been detected by chemical analysis, the proof of its administration by Thomson

⁴⁷ It is generally recognised that one of the founders of the modern science of toxicology was Mateo J. B. Orfila (1787-1853), born on the island of Minorca. He received his early education in Spain, and then settled in Paris where he studied medicine and chemistry. He received his degree in medicine from the University of Paris in 1811. He paid special attention to chemistry and physiology. In 1814 he published his two-volume work, *Traité de Toxicologie*, which is now a classic text and has gone through many editions. He was an experimentalist and developed a number of tests for identifying toxic agents. However, this was mere theory not founded on fact, and not one instance had ever been discovered by any medical man where prussic acid was found in a body, except where it was known to have been swallowed during life.

⁴⁸ "Orfila was of opinion that prussic acid might arise in the dead body from decomposition. He changed his opinions very frequently, and he never produced a single fact in support of that opinion. Dr Christison does not hold that opinion." - Cowan, S., *EMJ*, vol 3, 1857-1858, p945, Eaglesham Poisoning Case- Medical Evidence.

⁴⁹ The other two trials being that of Captain John Donellan in 1781 for the murder of Sir Thomas Broughton and the trial of John Talwell for the murder of Sarah Hart in 1845. See note 12.

⁵⁰ This was in relation to the amount of prussic acid found in the stomach where the evidence given by Dr Maclagan was not as strong as that given by the Doctors M'Kinlay. Further, whilst the Doctors M'Kinlay found no prussic acid in the spleen Dr Maclagan met with unequivocal evidence of the presence of prussic acid there. This however could have been due to the fact that as prussic acid is so very volatile it can soon escape from a body leaving detection very difficult.

was clear.⁵¹

The evidence led against Thomson at trial was so comprehensive that it was difficult for the jury to find any grounds upon which to consider an acquittal.⁵² From the evidence of witnesses it appears that, following discussions with neighbours regarding the trial of Madeleine Smith, Thomson developed an unhealthy academic interest in prussic acid.⁵³ Indeed, such was his interest that Thomson visited a photographer in Portugal Street, Glasgow, on July 10th 1857 to ask questions about prussic acid.⁵⁴ A painter named Armeil, who was present, told Thomson that prussic acid would have suited Miss Smith better than arsenic as it was not so easily discovered and could be easily obtained from any druggist.⁵⁵ Thomson listened attentively to this information

⁵¹ This included the suddenness of the attack of illness and the symptoms exhibited by Agnes Montgomery which were characteristic of poisoning by prussic acid soon after Thomson had left her room. Further was the odour of bitter almonds perceived in the room, fragments of a broken glass phial found immediately after Thomson left the garden to fetch Dr Scott, and a key that was found at the foot of a tree on the common, in which the prisoner, returning from fetching the doctor, appeared to have an unusual interest. The key was identified by the police as being a key to Agnes Montgomery's door. Record of the Lord Advocate AD14/57/123.

⁵² As it took Agnes Montgomery fifty minutes to die it is only fair to note that "cyanides are reputed to be lightning killers and that the active ingestion of prussic acid is calculated to cause immediate collapse and death within seconds. It is quite wrong, however, to assume that poisoning by cyanide must on all occasions result in rapid death. On the contrary, there is ample evidence that not infrequently collapse and death are delayed for at least sufficient time to permit volitional acts such as disposing of the remains of the poison and the container." - Polson, C.J., & Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London, 1973, p145. For evidence relating to delay of death see - Willcox, Sir William, *Med. Leg. Rev*, vol 2, 1934, pp332-336, In Discussion & Casper, J.L., *Handbook of Forensic Medicine*, Oxford University Press, London, 1862, p431. For cases where death has been almost instantaneous see R v Ball [1860] (Lewes Assizes) and the case of R v Tawell [1845] (Aylesbury Assizes) - Browne, G.L. & Stewart, C.G., *Reports of Trials for Murder by Poison*, Steven & Sons, London, 1883, pp16-52. As Agnes Montgomery was a large woman who was nearly 6ft in height, this may in part have accounted for the length of time it took her to die.

⁵³ Prior to the repeated acquisition of arsenic, Madeleine Smith had sent her father's page boy to a neighbouring chemist with a "line" asking the chemist to give the bearer half an ounce of prussic acid. The chemist declined to do so. Record of the Lord Advocate AD14/57/128.

⁵⁴ Prussic acid was extensively used in photographic processes.

⁵⁵ Record of the Lord Advocate AD14/57/123.

and on Thursday 12th September, one day before the murder of Agnes Montgomery⁵⁶, sent John Ferguson, a 15 year old stable boy, to Hugh Hart, druggist in Glasgow, to ask for sixpence worth of prussic acid.⁵⁷ On the 23rd September, Thomson sent Ferguson back to Hart to purchase another sixpence worth of prussic acid.⁵⁸ This was two days before the attempted poisoning of Mr and Mrs Mason. From the evidence given at the trial of Thomson by John Ferguson and George Stirling, shop assistants to Hugh Hart, there was unequivocal proof of purchase and possession of poison by the accused.⁵⁹

In addition, fragments of a glass phial, found in the garden shortly after Thomson had left the room of Agnes Montgomery, were presented in court.⁶⁰ With regards to the administration of prussic acid to Agnes Montgomery, Thomson must have emptied the contents of the medicine phial he possessed into the table beer she was drinking.⁶¹ As for the administration of prussic acid to the Masons, Thomson had every opportunity to add prussic acid to the whisky before arriving at their home.

⁵⁶ Thomson instructed the boy to tell the chemist that the prussic acid was for a portrait painter. In addition, Thomson told Ferguson that he wished the prussic acid to put on his hair to make it black and that Ferguson was to tell nobody of this purchase- Record of the Lord Advocate AD14/57/123.

⁵⁷ “Mr Hart, Please give the bearer 6d worth of prussic acid, John Thomson.” – Record of the Lord Advocate Ad14/57/123.

⁵⁸ Record of the Lord Advocate AD14/57/123.

⁵⁹ Thus satisfying the first principal circumstance on which reliance can be placed as establishing guilt or innocence.

⁶⁰ Throughout the trial the child, Janet Watson, had made various statements concerning the incident. In particular as to the possession of a medicine phial by Thomson which he wished rid of – “He took me to the back garden to get flowers. I saw him have a small bottle there. He played on it with his foot and broke it”. There was also the statement to her mother that the panel had promised her a “bawbee” not to tell of anything she had seen. To be noted, however, is that all statements given by 3 year old Janet Watson were held inadmissible. This was because the child had not told her mother anything of what had happened until 16 days after the incident, making any such statements not part of the res gestae. See O’Hara v Central SMT CO; 1941 S.C. 363; Teper v R [1952] A.C. 480. Further, of course is the fact that the child may have been considered too young to be reliable.

⁶¹ A Mrs Young who had attended Agnes when she took ill stated that she had found below the dresser in the room a bottle, containing about two gills of beer, which she had emptied out, as the bottle was required for hot water for the patient- Record of the Lord Advocate AD14/57/123.

Acquisition of the poison and the opportunity of secret administration by Thomson having been thus established, motive must be considered. Considering Agnes, Thomson had been most upset by her refusal to marry him and she had described him to relatives as “a liar and blackguard.” Further to this, on one occasion, Agnes had thrown water at Thomson after which he was heard to remark that “he would give it to her.”⁶² Antipathy between the two, and resentment of Agnes by Thomson, clearly existed. With reference to the Masons, motives are less easily attributable, though it is possible that Thomson was motivated by the desire to steal further from their household. There was precedent for this behaviour and it is possible that Thomson saw the Masons as a possible means of obtaining sufficient funds to move away from Glasgow.⁶³

Despite the differences in medical evidence for the prosecution, a unanimous verdict of ‘Guilty’ was returned by the jury on the murder charge, and Thomson sentenced to death. With regard to the charges for attempting to poison the Masons, the evidence was neither satisfactory nor conclusive during the trial.⁶⁴ Indeed, these two charges were treated as minor and perhaps only used by the prosecution to strengthen the murder charge.⁶⁵ Shortly after receiving his sentence, Thomson acknowledged his guilt and said that he was driven to commit the crime by an influence for which he could not account.⁶⁶ Thomson also confessed to having murdered a friend during his childhood by pushing him into a quarry-hole near his native village of Tarbert, Argyleshire.⁶⁷

⁶² Record of the Lord Advocate AD14/57/123.

⁶³ Thomson was perhaps worried following the death of Agnes Montgomery that her death would be traced to him.

⁶⁴ HMA v Thomson (1857) 2 Irvine 641. This was despite prussic acid having been detected in the bottle of whisky taken to the Mason household by Thomson.

⁶⁵ *Ibid*, p753.

⁶⁶ Record of the Lord Advocate AD14/57/123.

⁶⁷ Edinburgh Evening Courant, 29th December, 1857.

7.4 Conclusions

The trial of Thomson threw new light upon a poison which, from the apparent comparative rarity of its use, had not received the attention bestowed upon the investigation of other poisons during the nineteenth century. The question is therefore raised of how many offenders in the past escaped conviction due to the Lord Advocate and his deputes not being aware of how to master the complexities of medico-legal analysis for poisoning trials, and due to the medical inability to accurately diagnose and prove poisoning by prussic acid.

Killing with poison is often cast as a passionate, even romantic act and writers and novelists have commonly used poisons as murder weapons in their plots.⁶⁸ The same fascination with poisoning occurred during the early nineteenth century when details of murders were available in the form of halfpenny or penny broadsheets.⁶⁹ By then there had also begun to be a plethora of locally produced daily newspapers and it was even suggested that murder in the Victorian era, when so extravagantly publicised by the press, bred even more murder.⁷⁰ Indeed, it is very possible that Thomson was inspired by the trial of Madeleine Smith which began on the 30th June 1857.⁷¹ In

⁶⁸ For example in 1602 William Shakespeare had Hamlet's father killed with hebona (henbane) – See Act 1 scene V where Hamlet talks to his father's ghost. See also Macbeth, Act 1V, Scene 1, where in a cavern three witches are placing poisonous items into a boiling cauldron. In more modern times Alistair MacLean had his villains kill using aconite in horseradish in the novel *Bear Island*, Oxford University Press, London, 1976 and Dame Agatha Christie used poison in 83 different stories- See Gwilt, P.R. & Gwilt, J.R., *Pharm. J.* vol 221, 1978, p572, Poisoning with Plants.

⁶⁹ Altick, R.D., *Victorian Studies in Scarlet*, LM Dent & Sons Ltd, London, 1972, p44.

⁷⁰ “The publication in all their prurient and debasing details, of the foulest and bloodiest outrages ----- is invariably followed by a fresh crop of crime.” – Littell, J., *The Law Magazine*, vol v, 1845, p515, Littell’s Living Age.

⁷¹ “It happens, unfortunately, that great crimes, leading to the discussion which they must necessarily do, are often followed by the committal of the same offence on the part of others; and you will see how that comes out in a remarkable manner in this case.” – Charge of the Lord Justice Clerk, HMA v Thomson (1857) 2 Irvine 641.

particular, “even in the pretext alleged by Thomson to the stable boy for acquiring the poison we hear an echo of the cosmetic peculiarities of Blythswood Square.”⁷²

It would appear to me that the citation of only one prussic acid poisoning case in Scotland in this thesis is not an indication that poisoning with prussic acid was rare in the past. Given that prussic acid was freely available until the passing of the Pharmacy and Poisons Act of 1868, symptoms of poisoning with the acid could resemble epilepsy, and since forensic testing was imperfect and speculative, it is arguable that the temptation to use this poison occurred far more often than is recorded.⁷³

In this case, as in many of the others documented in the previous chapters, it is clear, that unlike the situation which pertains in the legal paradigm of the late-twentieth and early twenty-first centuries, scientific evidence was not accorded a special and more privileged position in Victorian trials. Critically for the Victorian mindset, as mentioned previously, poisoning was most convincingly shown if poison could be found in the body of the deceased. Though the conviction of Thomson was secured however, and medical evidence helped bring the case to trial and bolster a very strong indictment, great detail is lavished in the case upon the past behaviour and character of the accused and upon establishing the purchase of the poison and the potential motive for its administration. Circumstantial evidence is still relied upon to supplement the ‘proof’ of chemical analysis.

⁷² See Roughead, W, *Glengarry's Way and Other Stories*, W Green & Son Ltd, Edinburgh, 1912, p142. Thomson had said that he wished the prussic acid for use as a hair dye whilst Madeleine had procured arsenic in the pretext of improving her complexion.

⁷³ “A doctor once told me that he did not believe there was a single medical practitioner in London, of twenty years standing, who had not serious reason to believe that wives in his practice had poisoned their husbands and husbands their wives; but in the vast majority of cases the doctors could not utter their suspicions.” – Kellett, E.E., *As I Remember London*, Cassell & Co. Ltd, London, 1936, pp232-233.

APPENDIX 23

Prussic Acid Poisoning Cases

Date	Place	Accused	Victim(s)	Medium of Administration	Excuse for Obtaining Prussic Acid	What Happened to Victim(s)	Trial Verdict
1857	Eaglesham Glasgow	John Thomson	Agnes Montgomery, Agnes Mason, Archibald Mason	Table Beer and Whisky	Portrait Painting and Hair Colourant	Agnes Montgomery Died. Both Masons Recovered	Guilty-Executed

APPENDIX 24

Historical Tests Employed For Detecting Prussic Acid⁷⁴

1. Nitrate of Silver Test- Nitrate of silver yields with hydrocyanic acid a curdy white precipitate, the cyanuret of silver, which is so similar to the chloride of silver, that it is distinguished from it with difficulty. The precipitate is insoluble in nitric acid at room temperature, soluble in boiling concentrated nitric acid, very soluble in ammonia and not easily blackened by light. The precipitate is easily decomposed by the action of heat and free contact of air, so as to give cyanogen and metallic silver the former easily recognised by smell.
2. Sulphate of Copper Test- a few drops of sulphide of copper are mixed with the suspect liquid along with a slight excess of potash. If hydrocyanic acid is present a blue precipitate forms which is redissolved by the least possible excess of muriatic acid.
3. Persulphate of Iron Test- one drop of the suspect material mixed with two or three drops of alcohol and treated with sulphate of iron and potash will give a well marked precipitate of Prussian blue if hydrocyanic acid is present.

⁷⁴ Note

Tests 1, 3 - Taylor, Alfred, Swaine, *On Poisons in Relation to Medical Jurisprudence and Medicine*, 2nd edn, John Churchill, New Burlington Street, 1859, p668.

Test 5 – Polson, C.J., & Tattersall, R.N., *Clinical Toxicology*, (2nd edn), Pitman, London, 1973, p149.

Tests 2, 4 – Turner, M.D, “On the Tests of Prussic Acid”, *EMJ*, 1828, vol 30, p346.

Test 6 – Orfila, M.J.B. “On Prussic Acid”, *Lancet*, vol 1, 1828-1829, p737.

Test 7 – Polson, C.J., & Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London, 1963.

Test 8 – Glaister, John, *Medical Jurisprudence and Toxicology*, 12th edn, E & S Livingstone Ltd, London, 1962, p686.

Test 12 – Cowan, S, “Eaglesham Poisoning Case”, *EMJ*, vol 3, 1857-1858, p939.

Tests 9, 10, 13, 14, 16 – Autenreith, William, *Laboratory Manual for the Detection of Poisons & Powerful Drugs*, 5th edn, translated by Dr William H. Warren, P. Blakison's Son & Co., 1012 Walnut Street, Philadelphia, USA, 1921, pp21-25.

Tests 11, 15, 17, 18 are taken from Records of the Lord Advocate.

4. Proto-Sulphate Test or Common Green Vitriol Test- To a concentrated solution of green vitriol add successive portions of nitric acid, until the nearly black tint at first produced is converted into a red. Evaporate the solution to perfect dryness, dissolve the persulphate in water and filter. On mixing a little of this persulphate with the solution to be tested and adding pure potash a precipitate will form. The precipitate will dissolve in sulphuric acid with the solution presenting the yellow colour of the salt of iron without any Prussian blue being generated if prussic acid is present.
5. Smell- Prussic acid has a strong odour of peach-blossom, laurel water or bitter almonds. Noteworthy, is that not everyone has the ability to detect cyanide by smell.
6. Mix suspect material with coloured fluid so as to produce on the addition of nitrate of silver or persulphate of iron, brown precipitates, if hydrocyanic acid is present. Impregnate a piece of writing paper with a solution of caustic potash and then dip into the suspect fluid for three minutes. After drying the paper in air sprinkle a saturated solution of persulphate of iron over it. If hydrocyanic acid is present the paper immediately turns a blue colour, with a slight greenish hue.
7. Prussic acid vapour taints cigarette smoke and is thus detected by smoking as traces of vapour impart an unpleasant flavour to the tobacco.
8. Hydrocyanic acid is feebly acidic and reddens litmus paper slightly.
9. Copper-Quaiacum Test- Place a thin layer of suspect tissue or blood, acidified with tartaric acid, in a conical flask. Cork the flask and suspend in it by the cork a strip of filter paper soaked in a 10% solution of quaiacum resin, dried and then soaked in a 0.1% solution of copper sulphate in water. Dry and cut into strips. If hydrocyanic acid is present the paper will be turned a blue or greenish blue colour.
10. Thiocyanate Test- Add a solution of sodium, potassium sulphide or yellow ammonium sulphide drop by drop to the suspect solution until a yellow colour persists. Evaporate the mixture to dryness and then dissolve the residue in a little water acidified with dilute hydrochloric acid. A red colour produced by the addition of ferric chloride solution will indicate the presence of hydrocyanic acid in the original test solution.

- 11. Colour Tests** - Prussic acid will give an orange-yellow colour with dry sodium picrate, a greenish-blue colour with quaiacum and copper sulphate solution and a pink colour with phenolphthalein and copper sulphate in alkaline solution. The colours may be obtained by exposing the test papers impregnated with the various reagents.
- 12.** Add a drop of moderately dilute silver nitrate to the substance to be tested on a watch glass. Gently warm and if hydrocyanic acid is present the drop will become opaque-white from the formation of silver cyanide.
- 13. Uranium Test-** A grain or two of pure ferrous salt and the same quantity of uranium nitrate are dissolved in half an ounce of water. Place two or three drops of this on a white plate and add a drop of the suspect liquid. A purple precipitate or a greyish purple colour in weak solutions indicates the presence of hydrocyanic acid. Cobalt nitrate may be used instead of uranium salt, and is nearly as delicate.
- 14.** Neutralize suspect solution with alkali and mix with picric acid. A deep blood-red colour forms if prussic acid is present.
- 15.** Slightly alkalize suspect solutions with potash, add a few drops of cupric sulphate and then add just enough hydrochloric acid to dissolve the excess of cupric hydrate. If hydrocyanic acid is present white cuprous cyanide will remain undissolved.
- 16.** Mix suspect solution with excess alkali, add cobalt chloride and tartaric acid. On exposure to air a deep brown-red colour is produced if prussic acid is present.
- 17.** Add potash in excess then a little very finely, pulverised mercuric oxide. Mercuric oxide will dissolve as solution in alkaline fluid, only in the presence of hydrocyanic acid.
- 18.** Mercurous nitrate gives a black deposit of metallic mercury and a solution of mercuric cyanide in the presence of hydrocyanic acid.

APPENDIX 25

Post Mortem Appearances⁷⁵

1. Skin commonly livid, or tinged a violet colour
2. Head, face and lips violet in colour and bloated.
3. Frothy blood issuing from the nose and mouth.
4. Gut of a black colour.
5. Patches of redness on mucous membrane of stomach often with granular appearance owing to enlargement of mucous glands.
6. Kidneys a violet colour, softened and filled with blood.
7. Liver, spleen and kidneys gorged with black blood.
8. Veins of liver gorged with black blood.
9. Windpipe a bluish-black colour and filled with black blood.
10. Substance of heart firm.
11. Strong smell of bitter almonds.
12. Nails blue.
13. Liver dark in colour and smaller than usual.
14. Gall ducts contain violet coloured bile.
15. Lungs tuberculous.
16. Brain turgid.
17. Blood in every part of body with hydrocyanic odour.
18. Cyanosis of face.
19. Body surface with pink, irregular patches.
20. Face including lips of a reddish colour.

⁷⁵ **Note**

Aparances 1, 2, 3, 5, 6, 7, 9, 16, 21, 22, Taylor, Alfred, Swaine, *On Poisons in Relation to Medical Jurisprudence and Medicine*, 2nd edn, John Churchill, New Burlington Street, 1859, pp650-655.

Aparances 11, 18 – Polson, C.J., & Tattersall, R.N., *Clinical Toxicology*, 2nd edn, Pitman, London, 1973, p150.

Aparances 4, 8, 10, 12, 13, 14, 15, 17, 19, 20, 23, 24 – Cowan, S, “Eaglesham Poisoning Case – Medical Evidence”, *EMJ*, vol 3, 1857-1858, p938

Apperances 25, 26 are taken from Records of The Lord Advocate.

- 21.** Eyes bloodshot and pupils dilated.
- 22.** Appearance of asphyxia.
- 23.** Trunk of body swollen.
- 24.** Tongue swollen.
- 25.** Skin covering of a greenish-yellow colour.
- 26.** Spleen advanced in decomposition.

Chapter 8

Conclusion

8.1 A Hazardous Market for Poisons

The figure of sixty-three poisoning cases, spanning a period of one hundred and thirteen years, may suggest that the number of murders and attempted murders by poison in Scotland was comparatively small.¹ It would be extremely improbable to argue however, that these sixty-three cases represent accurately the amount of deaths by poison which may have occurred due to criminal intent. A successful murder by poisoning is one that is not recognised as a crime and the body buried without suspicions being roused.² The absolute certainty, facility and simplicity of operation by which most poisons may now be recognised stands out very starkly from the experimental procedures and lack of expertise which characterise toxicology a hundred years ago or two hundred years ago.

In the early nineteenth century there were also no controls over the sale of poisons in the United Kingdom. In the past, poisons were indiscriminately vended by the apothecary and then by chemists and druggists.³ Indeed, in this country any person,

¹ In England during 1750-1914, a period of one hundred and sixty-four years, there were 358 poison cases within the field of the six poisons in this work- See Watson, K., *Poisoned Lives, English Poisoners and Their Victims*, Hambledon and London, London, 2004. Further, in the Old Bailey during 1739-1878, a period of one hundred and thirty-seven years, there were 48 cases- See Forbes, T.R., *Surgeons at the Old Bailey*, Yale University Press, London, 1985, Table 8.

² In 1902 an Act of parliament was passed “For the Regulation of burning human remains, and to enable burial authorities to establish crematoria.”- The Cremation Society of Great Britain, *The History of Modern Cremation in Great Britain from 1847*, p67. The incineration of a body will obliterate many traces of crime unlike a burial where a body can be at a later stage exhumed and examined. If exhumation is required in Scotland, a sheriff, after petition, may grant a warrant. Certain precautions, however, have to be adopted in that exhumation should be made during the lightest part of the day, and when possible, the dissection conducted in the open – See Glaister, J., *Medical Jurisprudence and Toxicology*, 12th edn, E& S Livingstone Ltd, London, 1962, pp 37-38.

³ “Until the end of the 18th century, the practitioner of pharmacy in Great Britain was the apothecary. However, the apothecary did not stay by his laboratory: he became a prescriber as well as a supplier of drugs and by 1858 his position as a medical practitioner was recognised by registration under the Medical Act.” – Linstead, H., *Poisons Law*, Pharmaceutical Press, London, 1936, p19.

however uneducated or ignorant, could dispense medicines until 1843.⁴ Ignorance or carelessness on the part of a chemist or druggist, or actual prescription errors, could thus easily convert the healing draught of a physician into a speedy death-potion; such mistakes could perhaps have escaped detection. Further, as in the cases mentioned in Chapter Five, neglectful vending of poisons such as strychnine, was commonly held as an excusable error by juries.⁵

Pre-modern Scotland was racked with many acute and chronic diseases that all too commonly resulted in premature deaths. The populace when sick would eagerly buy whatever medicines were available.⁶ Often they purchased patent medicines which were unrealistically marketed as being able to cure a diverse variety of ailments.⁷ This unregulated market was dangerous in the extreme, with the indiscriminate sale of the

most deadly of poisons.⁸ With any poison easily accessible to the general public, accounts of cases of “poisoning by mistake” in the press were extraordinarily

⁴ The Pharmaceutical Society was established in 1841 and granted a charter of incorporation by Queen Victoria on February 18th, 1843. Membership of the Society was dependant upon whether a person had passed a qualifying examination and been admitted to a statutory register.

⁵ Logically all substances can be poisons and it is the right dose which differentiates a poison from a remedy. For example the foxglove plant is a source of digoxin, which increases the force of cardiac muscle contractions and is used for failing hearts. Dispensed in excess, however, in badly prepared decoctions in the past, or when, due to the similarity of the leaves being mistaken for comfrey, which when made into tea takes care of many digestive and stomach problems, would just bring on a fatal heart attack.

⁶ Common illnesses of the past, for which potentially dangerous medicines were available, included gout, worms, rickets, scurvy, cholera, diphtheria, pneumonia, smallpox, scarlatina, typhus, skin ulcerations and venereal disease.

⁷ Patent medicines became extremely popular in the latter half of the nineteenth century due to increased advertising in newspapers and magazines, expansion of transportation routes, low postal rates and increased literacy. The medicine manufacturers preyed on the publics’ fear of disease and nostrums were sold claiming to cure or prevent venereal disease, tuberculosis, cholera, scarlet fever, neuralgia and female complaints. Often these patent medicines contained poisons such as arsenic, mercury, laudanum, morphine, strychnine and cocaine.

⁸ From 1769 until 1869 one of the most influential medical books in Scotland was William Buchanan’s *Domestic Medicine*, which recommended that every well- equipped home should have supplies of such

common.⁹ The multitude of accidental poisonings which arose from lack of regulation increased the possibility of deliberate cases of poisoning passing undetected. Indeed, the safety and well being of society at large imperatively demanded the imposition, by government, of stringent restrictions upon the indiscriminate sale of deadly drugs. Britain seems to have lagged far behind other countries in relation to controlling the sale of poisons, in particular, France, Germany, Russia, Italy and Prussia.¹⁰ In fact as far back as 1365 Italian laws made the sale of certain poisons illegal, unless the person to whom they were sold was not only an adult, but was also known to the apothecary.

Under-regulation is attributable to the desire not to restrict free trade in poisons and articles containing them. In particular, this was the case for opium, which was imported in huge quantities from Turkey during the nineteenth century, and then sold off by way of auctions, drug brokers and private arrangement.¹¹ As another example, arsenic, was not only used in fly papers, rat poisons and medicines, but extensively employed as a colouring agent in curtains, furniture fabrics, lampshades, ornaments, artificial flowers, carpets, linoleum, children's toys, clothing and candles during the Victorian era.¹² In fact, it is probable that no Victorian household in Britain would

drugs as: Elixir of vitriol, Flowers of sulphur, Genitian root, Liquid laudanum, Nitre, Oil of almonds, Jalap, Pennyroyal water, Snake root, Sal ammoniac, Sweet spirits of nitre, Syrup of poppies, Tamarind, Yellow basilicum root etc. Indeed, it was claimed that every Scottish cottage had a copy of Buchan's *Domestic Medicine* and the Bible. See Porter, R., *Quacks, Fakers and Charlatans in English Medicine*, Tempus Publishing Ltd, 2000, pp47-49.

⁹ "Not a week passes, but we read in the public prints of cases of "poisoning by mistake," and the usual regret is expressed, that the sales of such dangerous substances should be transacted with so little supervision on the part of the authorities." – Cook, W.S., *EMJ*, vol 2, 1856-1857, p750, The Sale of Poisons.

¹⁰ See Cook, W.S., *EMJ*, vol 2, 1856-1857, pp764-767, The Sale of Poisons; Burney, I., *Journal of British Studies*, vol 38, 1999, pp59-92, Poisoning of no Substance: The Trials of Medico-Legal Proof in Mid-Victorian England.

¹¹ Berridge, V., *Opium and the People*, Free Association Books Ltd, London, 1999, pp3-7. Note that in 1872 the consumption of opium in Britain was 17,000 lbs whilst by 1853 this figure had risen to 67,000 lbs – Parliamentary Papers: *Annual Statistics of Trade, Imports and Exports of Opium 1827-1860*.

¹² Burney, I., *Wellcome News*, vol 20, 1999, p3, The Poison Hunter.

have been free from the presence of arsenic.¹³ This is in contrast to other European countries where the use of arsenic was prohibited in many household goods.¹⁴ Further, to the above was the huge British trade in phosphorus matches from the 1830s until 1910, when such matches were outlawed.¹⁵ Governments throughout the Victorian period put the maximisation of profit through *laissez-faire* policy above concerns for the safety of the population.

There were no effective legislative controls over the sale of poisons until the introduction of the Arsenic Act of 1851. This arose due to increased parliamentary pressure following an increase in the number of deaths from poisoning, of which more than one-third of said deaths were due to arsenic poisoning.¹⁶ The next main piece of legislation was the Pharmacy and Poisons Act of 1868 which restricted the selling of poisons to sales by doctors, pharmacists and registered druggists, and listed in two separate schedules certain substances to be considered poisons. It did not, however, cover all poisons. This Act remained substantially unchanged until the Pharmacy and Poisons Act of 1908 when tighter controls over the sale of opium products and carbolic acid were introduced, particularly as carbolic acid was a disinfectant favoured by suicides. Apart from “Dangerous Drugs” legislation no further amendments were made regarding the sale of poisons until the Pharmacy and Poisons Act of 1933.

8.2 Conditions For A Mid-Century Surge in Poisoning

¹³ See Galent, D., *Gamages General Catalogue, Yesterday's Shopping*, Wordsworth, Hertfordshire, 1994. For example in the Gamages General catalogue of 1875 a child's model open touring car could be purchased with a torpedo body and superior quality hand painting involving arsenical paint for 11/9; a maids' sports coat which would have contained arsenical dyes could be bought for 12/11 and a candle shade with gimp top and base with beaded fringe dyed with arsenic could be purchased for 9d.

¹⁴ Ryan, M., *EMJ*, vol 1, 1845, p380, The Use of Arsenic in Household Goods.

¹⁵ For example, during the late 1800s Bryant and May were making sixty billion matches containing phosphorous a year- Emsley, J., *The Shocking History of Phosphorous: A Biography of the Devil's Element*, Macmillan, London, 2000.

¹⁶ Linstead, H., *Poisons Law*, The Pharmaceutical Press, London, 1936, p3.

In his book, “Poisons Law”, Linstead states that “(P)harmacy and poisons legislation in Great Britain has developed according to no logical scheme” and that “it is rather the product of expediency, and its present form is largely due to the casual nature of its early growth.”¹⁷ According to the cases presented in the preceding chapters it would appear that poisoning crimes in Scotland reached a peak between the 1840s to the 1860s.¹⁸ Forty percent (40%) of all the cases occurred over these three decades¹⁹ with arsenic cases making up 22% of this figure.²⁰ English poisoning cases also reached a peak during the mid nineteenth century with the middle decades of the nineteenth century being considered a high point in English criminal poisoning.²¹ The crime of poisoning in England had grown significantly over the first half of the decade²² with newspapers such as the Illustrated Times declaring that criminal poisoning was the “crime of the age” and “the crime of civilization.”²³ Indeed, it has even been suggested that to take heed against poison was one of the waking thoughts common to all during the mid half of the twentieth century.”²⁴ Factors such as the movement of people from rural locations to cities, increases in population, worsening income gaps between the rich and the poor due to industrial development, increase in rate of detection and lack of legislation controlling the sale of poisons, all contributed to this mid-century surge. Moreover as Bartrip points out, “at a time when divorce

¹⁷ Ibid, p1.

¹⁸ “The 1850s were the era of the high-profile poisoner. A series of celebrated murder trials introduced an ever anxious public to the terrors of the slow, the sophisticated – indeed the scientific poisoner, and to his nemesis, the intrepid poison hunter. With a fervid press watching every move, the fear of poison drove changes in law and medicine.” - Burney, I., *Wellcome News*, vol 20, 1999, p3, The Poison Hunter.

¹⁹ $25/63 \times 100 = 40\%$

²⁰ $14/63 \times 100 = 22\%$.

²¹ See Burney, I., *Poison Detection and the Victorian Imagination*, Manchester University Press, 2006, p12. See also Bartrip, P., *Medical History*, vol 36, 1992, A Pennurth of Arsenic for Rat Poison: The Arsenic Act, 1851 and the Prevention of Secret Poisoning.

²² The number of trials for murder by poison during the 1830s was triple the number of trials for 1810, then rose by more than 50% the following decade- See Whorton, J. C., *The Arsenic Century: How Victorian Britain was Poisoned at Home, Work and Play*, Oxford University Press, 2010, p1.

²³ Burney, I., *Poison Detection and the Victorian Imagination*, Manchester University Press, 2006, p12. The Illustrated Times was a cheap illustrated newspaper launched in Fleet Street in London in 1855.

²⁴ Whorton, J.C., *The Arsenic Century: How Victorian Britain was Poisoned at Home, Work and Play*, Oxford University Press, 2010, p1.

was wellnigh unattainable, it offered a temptingly quick escape from an unhappy marriage.”²⁵

Further, during the 1840s, industry came to a standstill and the country experienced mass unemployment leaving many living in extreme poverty and desperate. There began to be a rise in the insurance industry and burial clubs, where for a small weekly payment such as a penny, a lump sum to cover a funeral would be paid upon death.²⁶ In particular, the lives of many children born into abject poverty were insured and often such profits were further maximised by the joining of several clubs at the same time.²⁷ Membership rolls for such clubs were even referred to as “catalogues of the doomed” and the Death Clubs as they were known came to be widely regarded as the “prolific mother” of arsenical murder.²⁸ This period also witnessed a growing commerce in cadavers.²⁹ By this time also “scientific knowledge” had become more publicly accessible; education and literary rates increased, the press flourished and novels – described as “veritable handbooks for poisoners” – became widely available.³⁰ With the government failing to pass legislation until the Pharmacy and Poisons Act of 1868 the employment of poison was an ever-present temptation in people’s lives.³¹

²⁵ Bartrip, P., *English Historical Review* 1994, vol 109(433), p893, How Green Was My Valance? Environmental Arsenic Poisoning and the Victorian Domestic Ideal.

²⁶ See Tennyson, *Maud*, - “When a Mammonite mother kills her babe for a burial fee, And Timour-Mammon grins on, a pile of childrens’ bones.” – Tennyson, Alfred, Lord, *Poetical Works*, Macmillan & Co Ltd, New York, 1899, Part 1, X11, Lines 45 & 46.

²⁷ Whorton, J.C., *The Arsenic Century: How Victorian Britain was Poisoned at Home, Work and Play*, Oxford University Press, 2010, p30.

²⁸ Ibid.

²⁹ See Adams, N., *Scottish Body Snatchers; True Accounts*, Goblinshead, Musselburgh, 2002. See also Roughead, W., *Trial of Burke & Hare*, Notable Scottish Trials, W. Hodge & Co Ltd, Glasgow & Edinburgh, 1921.

³⁰ Burney, I., *Wellcome News*, vol 20, 1999, The Poison Hunter, p57.

³¹ Note that there was however the Arsenic Act of 1851.

This surge in Scottish cases confirms the arguments made by Burney³² and Golan³³ for widespread interest in refining both the medical and legal pursuit of poisoners in the mid-Victorian period. Burney writes that (H)istorians of medieval homicide have argued that poisoning cases and ‘stealth’ modes of killing generally, were comparatively rare” and further more that “(I)t is not until the middle decades of the nineteenth century- especially the 1840s and early 1850s- that the situation seems to have changed markedly....there is good reason to consider the middle decades of the nineteenth century as a high-point of criminal poisoning.”³⁴

8.3 The Development and Limits of Forensic Medicine

The use of poison was further facilitated by the fact that until the beginning of the 1800s, medical jurisprudence was in its infancy in this country.³⁵ There was no treatise dealing with forensic medicine, excepting a small book published in 1788 by Samuel Farr.³⁶ Indeed, study of forensic medicine was not pursued as a separate science, and it found no place in the teachings of medical schools. Any knowledge possessed by members of the medical profession was imperfect, and such as there was had to be borrowed almost entirely from foreign sources. Commentators of the day lamented the weak position of this science as a recognised field of knowledge and

³² Burney, I., *Journal of British Studies* 1999, 38, pp59-92, A Poisoning of No Substance: The Trials of Medico-Legal Proof in Mid-Victorian England; Burney, I., *Studies in History and Philosophy of Science* 2002, 33(2), pp289-314, Testing Testimony: Toxicology and the Law of Evidence in Early Nineteenth Century England.

³³ Golan, T., *Laws of Men and Laws of Nature: The History of Scientific Expert Testimony in England and America*, Cambridge MA: Harvard University Press, 2004.

³⁴ Burney, I., *Poison Detection and the Victorian Imagination*, Oxford, Blackwell, 2006, pp19-20.

³⁵ This is in contrast to the continent where in Germany, France and Italy for example, numerous workers had been engaged for many years, during the 18th century onwards, in the study of medical jurisprudence and it had assumed the position of a separate and important branch of medicine.

³⁶ The book was entitled “*The Elements of Medical Jurisprudence*”, but was merely an abridgement of an old work by Fazelius, a Geneva professor – See Maclagan, C., *Journal of Jurisprudence*, vol XXV, 1881, p620, Medical Jurisprudence: Address on the Occasion of the Opening of the Courses of Law in the University of Edinburgh.

practice in Britain.³⁷ It was a subject in which there was an immense lack of awareness and ability during the early decades of the nineteenth century.

Many cases of poisoning would just not have been recognised at all by the medical profession in Scotland during the early nineteenth century. From 1807 until the early 1830s, the University of Edinburgh was the only medical school in Britain to give lectures in forensic medicine.³⁸ These lectures did not become compulsory, however, until 1833, and in any case did little to develop a well ordered study of medical jurisprudence as applied to the criminal courtroom. Glasgow University did not introduce forensic medicine as a compulsory part of medical examinations until 1839.³⁹ That Edinburgh was the only university in Britain to give instruction in forensic medicine until the 1830s serves only to further highlight the huge gaps which existed in medical knowledge throughout the country and the potential for error which therefore existed in legal cases. Further, it was not until the second decade of the nineteenth century that a systematic work on the subject was published – earlier works being little more than selective glosses on a continental treatise tradition already well established by the sixteenth century.⁴⁰

Courts of law, naturally, were often compelled to depend upon the medical practitioner for primary information. If the medical practitioner had not had due training to regard medical questions in a forensic light, it is hardly surprising that he might be apt to miss important points during any examination, or, indeed, to make

³⁷ Burney, I., *Poison Detection and the Victorian Imagination*, Manchester University Press, 2006, pp40-41.

³⁸ See Crowther A & White, B., *On Soul and Conscience: The Medical Expert and Crime: 150 years of Forensic Medicine in Glasgow*, Aberdeen University Press, 1988, p7.

³⁹ Ibid. Note that the first lectures at Edinburgh University were given by Andrew Duncan, Professor of the Institutes of Medicine at Edinburgh who saw the benefits of the use of forensic medicine for the detection of crime.

⁴⁰ Burney, I., *Poison Detection and the Victorian Imagination*, Manchester University Press, 2006, p41.

mistakes which may have led to miscarriages of justice.⁴¹

In order to give judgement in cases of poisoning, the medical jurist should be acquainted with many factors. These include the different poisons, their physical and chemical characteristics, their effects on the human body and the means of distinguishing them from all substances with which they may be confused or by which they may be obscured. The medical jurist had to act as a mediator between the insensible and the sensible with the task of demonstrating the presence of things not evident to others.⁴² Unfortunately, however, at most medical schools in the nineteenth century, the subject of forensic medicine was looked upon as being of comparatively little importance. Indeed, generally the youngest and least experienced pathologist was selected to discharge the duties that should have been undertaken by the Professor. Having never dissected a corpse in an important medico-legal case, or given evidence in a court of law, his lectures would lack that practical stamp with which to command the attention of his audience. Further, such annual courses were often taught in a listless and unsatisfactory manner. It is of little surprise therefore that medical students of the past showed merely a superficial acquaintance with the subject of medical jurisprudence, or that medical evidence was generally distrusted by the courts, owing to the hesitation and unsatisfactory knowledge displayed by medical men in the witness box.⁴³

⁴¹ This was particularly the case in smaller communities where the general practitioner was the only available expert unlike Glasgow and Edinburgh where the fiscal would usually call an experienced police surgeon for post-mortem and a reputable chemist for toxicological analysis- See Crowther, A & White, B., *On Soul and Conscience: The Medical Expert and Crime: 150 Years of Forensic medicine in Glasgow*; Aberdeen University Press, 1988, p19.1

⁴² Ibid, p6.

⁴³ See Taylor, A., *EMJ*, vol 14, 1868-1869, p745, The Principles and Practice of Medical Jurisprudence – “A medical jurist should have a theoretical and practical knowledge of all branches of the profession, a large range of experience, and the rare power of adapting his experience to emergencies. He should be able to elucidate and difficult medico-legal questions which may arise, and be prepared at all times to make a cautious selection of such medical facts, and a proper application of such medical principles, as may be necessary to enable a judge to place the subject in an intelligible light before the jury and to enable a jury to arrive at a just decision.”

Indeed, the important requirement, in any suspicious case, of the preliminary examination of the corpus delicti being entrusted to medical men of experience was not met.⁴⁴ Errors could therefore easily be made, cases of poisoning not be recognised and there often be doubt as to cause of death. The medical facts recorded during preliminary examinations into cause of death during the early nineteenth century would frequently, therefore, fall short of what was required for an effective trial in Scotland. Such investigations were inadequate and indeed sometimes non-existent in cases of sudden death. The medical profession had very little interest in medico-legal science during the early nineteenth century and it is therefore possible that many poisoning incidents were simply ignored.

Further, it was often difficult to distinguish between poisoning and many diseases. Errors arose from the similarity of poison symptoms and those of naturally occurring afflictions.⁴⁵ It is credible, therefore, that many persons who had been poisoned died after having been diagnosed and treated for a common illness. Whilst post-mortem examination might strengthen the evidence of poisoning, it was often unreliable except in cases of poisoning with the strong mineral acids.⁴⁶ In addition, some poisons occasionally disappear altogether from the stomach, as, for instance, preparations of opium and hydrocyanic acid.⁴⁷ Further, the decomposition of the body after burial

⁴⁴ Corpus Delicti being – did the person die of medical disease or poison? Note that in 1824 a code of instructions was drawn up by the Lord Advocate, and issued from the Crown Office to every procurator fiscal furnishing detailed directions to medical men for the making of post-mortem examinations – Maclagan, D., *The Journal of Jurisprudence*, vol XXII, p17, Forensic Medicine from a Scottish Point of View.

⁴⁵ Such diseases included plague, typhus, fever, apoplexy, cholera, inflammation of bowels and stomach, tetanus, strangulated hernia, haematemesis (vomiting of blood) and epileptic convulsions. Note that as recently as 1949 an English woman, Margery Radford, was diagnosed as having died from tuberculosis, when the real reason was that she had been poisoned by her husband with arsenic – see Simpson, K., *Forty Years of Murder*, Harper Collins, London, 1978, pp268-279.

⁴⁶ See Maclagan, D., *The Journal of Jurisprudence*, vol XXII, 1879, p18, Forensic Medicine from a Scottish Point of View.

⁴⁷ Simpson, K., *Taylor's Principles and Practices Medical Jurisprudence and Toxicology*, 12th edn, London, Churchill, 1965, p510.

prevents many, but not all, poisons from being detected.⁴⁸ Along with these difficulties, the medical profession was compelled to combat the widespread belief in the nineteenth century, which was shared by jurors, that no man can die of poison except if poison be found in his body. Given this bias, it is not surprising that relatively few criminals could possibly be brought to justice in poisoning cases, even given medical testimony.

The path of murder by poison was easy in the past, facilitated by medical ignorance. In the past many medical men had no previous chemical experience of testing for poisons, but carried out textbook tests anyway. By placing blind reliance upon such results, the accuracy of which could not be determined, or their value judged, by courts or juries, there was a great risk that a person might be acquitted on erroneous evidence. It is possible therefore that such a belief led to the failure to punish many guilty of the crime of poisoning.

Today, in contrast to the conditions of the nineteenth century, the chemical evidence of the presence of poison will normally be clear, distinct, conclusive and usually satisfactory.⁴⁹ In the Victorian period, several tests had to be carried out to be certain of the presence of arsenic, whilst both opium and strychnine broke down naturally in the body after a very short time.⁵⁰ Further, many medical experts engaged by the prosecution for poisoning trials in the past received but scanty justice. They were roughly handled in the witness box, and the testimony they gave was openly criticised in British medical journals and books.⁵¹ Indeed, in the 1860s there was great concern

⁴⁸ Arsenic has been detected months, years and even centuries after the interment of a person poisoned- Ibid. See also Weider, B & Hapgood, D., *The Murder of Napoleon*, Corgi Books, Transworld Publishers, 1982, pp72-74.

⁴⁹ The modern toxicologist has access to chromatography, microscopy, electrophoresis, ultra-violet Spectrophotometers, infra-red spectroscopy, X-ray diffraction, electron microscopy, measurements of Enzyme activity, the use of radioactive isotopes and biological assay – Curry, A. S., *Forensic Science Society Journal*, vol 1, 1960, pp91-96, Homicidal poisoning.

⁵⁰ See Crowther, A and White, B., *On Soul and Conscience; The Medical Expert and Crime: 150 Years of Forensic Medicine in Glasgow*, Aberdeen University Press, 1988, p20.

⁵¹ In particular, see the English trial of William Palmer for the poisoning of John Parsons Cook with antimony at Rugley in 1856- Brown, G & Stewart, C.G., *Reports of Trials for Murder By Poisoning*, Steven & Sons, London, 1883, pp84-232.

among British scientists about the role of the expert witness, particularly with regard to the effect of the legal constraints on the validity of scientific evidence.⁵² It was argued that to put a scientist in the position of an advocate was “far removed from the idea of a man of science.”⁵³ In accordance with these views and in particular the lack of any legal training it is very possible that many cases of poisoning were undetected or that often justice was frustrated.

Chemical tests were first admitted as evidence in 1752 in the English trial of Mary Blandy, accused of poisoning her father with arsenic.⁵⁴ The main test used here, however, was the throwing of the suspected powder on a red-hot iron with charcoal to see whether it would give off thick white fumes and the stench of garlic, indicating the presence of arsenic.⁵⁵ Indeed, until 1836 this was the prevalent chemical test for arsenic in suspected cases of poisoning.⁵⁶ Since arsenic appears to have been the poison of choice until the Arsenic Act of 1851, such a test hardly seems adequate in nature. Clear and satisfactory evidence required to be presented to a jury in any poisoning trial. Undoubtedly, this did not happen in the past and chemical analysis alone could not be relied on to supply the conclusive evidence of deliberate poisoning required to secure a criminal conviction.

The importance of being able to detect small quantities of arsenic, or indeed any poison, in cases where there was reason to suspect that it had been employed with the intention of destroying life, is obvious. Chemical testing was, however, presumptive and inadequate in the past, despite tests being described in many chemistry books. In addition, it was often the case during judicial investigations that medical men were obliged to perform experiments on very small quantities of a substance accidentally left

⁵² Coley, N.G., *Medical History*, vol 35, 1991, p414, Alfred Swaine Taylor, MD, (1806-1880): Forensic Toxicologist.

⁵³ Smith, R.A., *Journal Royal Society of Arts*, vol 7, 1860, p137, Medical Jurisprudence.

⁵⁴ See Roughead, W., *Trial of Mary Blandy*, Notable British Trials, W Hodge & Co Ltd, London, 1914. See also Glaister, J., *Surge Glasgow University Med J.J*, vol 26, pp82-88, 1952, The Poisoner: Some Aspects of Famous Trials.

⁵⁵ Campbell, W.A., *Medical History*, vol 25, 1981, p202, History of the Chemical Detection of Poisons.

⁵⁶ Some precipitation reactions were also used, but these did not apply only to arsenic eg: adding into a suspected solution sal ammoniac; lixivium of tartar, spirit of vitriol, spirit of salt or syrup of violets.

in a cup or bottle, or in the contents of the stomach. Arsenic, in particular was difficult to detect in very minute quantities, particularly given the lack of skill on the part of many doctors and chemists.

In 1836 James Marsh published his new reduction test for the detection of arsenic. Marsh claimed that he could detect as little as 1/120 of a grain of arsenic compound.⁵⁷ The Marsh test, however, was not very delicate and often gave rise to erroneous results.⁵⁸ It also required great technical skill and experience and it is highly likely that the procedures of the test were incorrectly carried out on occasion, leading to the possibility of wrongful verdicts being reached.⁵⁹ Noteworthy, is that Taylor was not happy with the Marsh process and suggested the test should only be used for corroboration purposes.⁶⁰ Reinsch's test, published in 1841, also received great criticism and in particular, during the Smethurst poisoning trial in England in 1859.⁶¹ Reinsch's procedure only partially contributed to improving testing accuracy and indeed the analyst was encouraged to carry out as many tests as possible, a most laborious business, as he could not trust any one test.⁶² The chemical tests used for arsenic were, therefore, neither decisive nor conclusive of its presence. This was also the case for the chemical tests used to detect other poisons. No specific tests – tests

⁵⁷ He claimed that he was able to “separate very minute quantities of arsenic from soup, gruel, porter and other alimentary liquors.” – Marsh, J., *Edinburgh New Phil Journal*, vol 21, 1836, pp229-236, 1836, Account of a Method of Separating Small Quantities of Arsenic from Substances with which it May be Mixed.

⁵⁸ See Crowther, A & White, B., *On Soul and Conscience: The Medical Expert and Crime: 150 Years of Forensic Medicine in Glasgow*, Aberdeen University Press, 1988, p20.

⁵⁹ See Gamgee, A., *EMJ*, vol 10, 1864-1865, pp408-415, On an Alleged Fallacy in Marsh's Process for the Detection of Arsenic.

⁶⁰ See Taylor, A., *Guy's Hospital Reports*, 3rd series, vol V1, 1860, p6, p71, p201, Facts and Fallacies Connected with the Research for Arsenic and Antimony, with Suggestions for a Method of Separating these Poisons from Organic Matter.

⁶¹ Dr Smethurst was tried at the Old Bailey in July 1859 for the murder of Isabella Banks, whom he had bigamously married, with arsenic. Although, Smethurst was found guilty by the jury and sentenced to death, the evidence was vitiated by the medical fuss and Smethurst reprieved. His murder conviction was thus quashed due to the scientific controversy surrounding the case. See Watson, K., *Poisoned Lives: English Poisoners and their Victims*, Hamledon & London, London, 2004, p208.

⁶² Crowther, A and White, B., *On Soul and Conscience: The Medical Expert and Crime: 150 Years of Forensic Medicine in Glasgow*, Aberdeen University Press, 1988, p20.

that applied solely to individual poisons- existed for the other poisons considered in this thesis namely opium, strychnine, phosphorous, prussic acid and corrosive acids.

Thus, there was no means of detecting opium in the past except through smell. Strychnine was too rapidly metabolised in the body; many organic compounds could inhibit the luminosity of phosphorous and prussic acid was so volatile that after a short time it was generally impossible to detect. Chemical testing could also not prove the presence in the body of corrosives, although chemical evidence could often be obtained by the application of various tests to clothing. Indeed, Taylor himself recognised that chemical analysis, no matter how carefully conducted, only provided a certain degree of probability and could never achieve the incontrovertible demonstrations of proof required by the courts.⁶³

8.4 Final Verdicts

It is of little surprise, therefore, that during poisoning trials of the nineteenth century, courts would view the results of chemical tests with some doubt. Chemical investigation in suspected cases of poisoning in the past was both unsatisfactory and could produce misleading results. During the nineteenth century chemical testing was still in its infancy and took many years to become fully accepted as evidence in court. Whilst analytical results were allowed in evidence, this was only to supplement medical observations and evidence from chemical analysis was always open to criticism and seldom if ever considered conclusive.⁶⁴

It is also possible that wrongful verdicts were reached in some cases and that, in the vast majority of instances of criminal poisoning, the plethora of alternative explanations for sudden death meant that cases simply did not proceed to trial. Even at trial, the improbabilities and inaccuracies of chemical testing and the complex and disputed results of chemical processes make it likely that lay juries of the past reached wrongful decisions.

⁶³ Coley, N.G., *Medical History*, vol 35, 1991, p414, Alfred Swaine Taylor, MD, FRS.

⁶⁴ Ibid, pp414-415.

Juries were certainly hindered by the great variability of the medical witnesses called and although medical testimony became more and more commonplace during the nineteenth century, the quality was often a hit and miss affair, very much dependent upon chance.⁶⁵ As Forbes writes: “(T)ime after time it was the expert witness, learned or ignorant in forensic medicine, careful or perfunctory, concerned or casual, who in effect determined whether the accused perished on the gallows, was transported for forced labour in the colonies, or went free.”⁶⁶ When the medical profession could not fully appreciate the subject of medical jurisprudence it is difficult to understand how a jury were meant to comprehend the importance of the scientific evidence they heard in a court of law and to know what weight of trust to give to medical testimony. Indeed, ignorant of the modern progress of science it is possible that jurors were astounded by the evidence given by experts in the courts.⁶⁷

Victorian juries knew little of the progress of chemistry, physiology and pathology and would regard as something marvellous, those tests and facts which well-educated men thought to be only ordinary scientific knowledge.⁶⁸ In particular, this was the case for evidence brought in a trial for murder by poison.⁶⁹ Upon the evidence before them in any such trial, many jurors would have undoubtedly experienced confusion. Jurors in Scotland do not have the advantage of hearing preliminary speeches by the

⁶⁵ Some medical practitioners had no idea of what to look for during a post-mortem, while others were vigorous and competent. In an unbroken link with medieval standards some were still prepared to state the cause of death without the benefit of a post-mortem. Even when a post-mortem was carried out often this was on a table in the house where the victim had died with family members and friends milling about. For example during the post-mortem on his final victim, William Palmer stood close to the table, contriving to jostle the surgeons so that some of the stomach contents were spilled, and even attempted to tamper with the sealed jar in which the organs were placed – Browne, G.L. & Stewart, C.G., *Reports of Trials for Murder by Poisoning*, Steven & Sons, London, 1883, pp107-109.

⁶⁶ Forbes, T.R., *Surgeons at the Old Bailey*, Yale University Press, London, 1985, pp107-109.

⁶⁷ The writer is of the belief that in many complex, modern day cases this situation still prevails.

⁶⁸ “It was not uncommon in the past for expert witnesses at trials to arrive with glass tubes containing arsenical mirrors, slips of copper stained with metallic arsenic, and samples of the coloured precipitates known to be formed by arsenic and other metallic poisons.” – Watson, K., *Poisoned Lives:English Poisoners and their Victims*, Hambledon & London, London, 2004, p19.

⁶⁹ Chemical results, physiological phenomena and pathological appearance would confuse the jury of the past and today.

Crown and counsel for the defence as was the case in England until 1848.⁷⁰ They, therefore, receive no introduction other than what is contained in the indictment, namely the bare facts and the nature of the offence charged. This means that during the initial stages of a trial, a jury may have difficulty in even understanding the relevance of the specialist evidence given by witnesses. This would have been particularly the case in the past when due to the lack of medico-legal experts, juries often had to depend upon the evidence of the medical practitioner, rather than an expert, for the primary information which would have been essential for their guidance in any poisoning trial.

Thus, undue importance was often attached to the actual finding of poison in an article of food or any one of the discharges from the body or in a dead body itself. Whilst there can be no doubt that this is an important link in the chain of proof, it is not an essential link as some poisons such as prussic acid are extremely volatile and can disappear within days. Further, some illnesses of the past, such as Asiatic cholera so closely mimicked the symptoms of arsenic poisoning that defence counsel in poisoning trials would use this as their client's defence.⁷¹ These matters, however, only served to make matters more perplexing for juries of the past.

In this respect the findings of the Scottish cases investigated and the tentative embrace of toxicological development by courts and juries enables us to reconsider the arguments of Watson on the importance of the increasing prevalence of the medico-legal expert witness.⁷² Watson writes that:

"Criminal poisoning fitted neatly into (the) area of overlap between science, medicine and the law because of the clear difficulties that proving a crime that was so often hidden from view (or presumptive) posed to a legal system that desired certainty. An increasingly demanding burden of proof, grounded in evolving rules of evidence,

⁷⁰ Summary Proceedings Act 5 & 6 Vict. ch. 89.

⁷¹ See Mariner, B., *Murder with Venom*, Pan Books, MacKay's of Chatham Ltd, Kent, 2003.

⁷² This is also asserted in Landman, S., *Law and History Review*, 1998, 16, pp445-494, One Hundred Years of Rectitude: Medical Witnesses at the Old Bailey, 1717-1817; Burney, I., *Studies in History and Philosophy of Science*, 2002, 33(2), pp289-314, Testing testimony: Toxicology and the Law of Evidence in Early Nineteenth Century England.

placed more and more emphasis on what legal commentators referred to as generally as the “medical testimony” in cases of suspected poisoning.”⁷³

The Scottish cases present a more complex picture of the development of medical witnesses. Taking into consideration the climate both of promotion and criticism of medical witnesses in the nineteenth century, given the failure to reach positive verdicts in many of the trials under consideration, it is possible to argue that though more emphasis may have gradually been accorded medical witnesses, they remained largely ineffective in Scotland. The option for juries to declare a case ‘Not Proven’ was still prevalent even in trials with multiple layers of medical and chemical evidence. While enthusiasm for scientific proof undoubtedly grew during the period in question, for many reasons already outlined, the ability of medical witnesses to provide sufficiently concrete evidence to overcome the possibility of a ‘Not Proven’ verdict was often lacking. The Scottish law in contrast to the commentary by Watson (and others such as Burney) placed an extra demand in terms of the character of proof required of forensic medicine. Science implicitly has come to represent concrete proof, but juries in the nineteenth century in Scotland only attributed circumstantial weight to scientific testimony.

In accord with Shapiro’s⁷⁴ notion of the development of circumstantial evidence in British law from the experience of dealing with the stealth crimes of witchcraft (particularly in the seventeenth century) and poisoning, it may be argued that, contra Watson, poisoning was only partially relevant to the new sciences of toxicology. While toxicology threw new light on the secrets of poisoning, law in England and Scotland had become accustomed to treating such cases in terms of building up catalogues of circumstantial data to inform a jury as to the background, logistical possibility and motive for crime, as well as concerning the character of the accused and victim. Thus, such reliance on circumstantial data remained, even after the revolutionary promise of toxicology had made its influence felt.

⁷³ Watson, K., *Medical History* 2006, vol 50, p378, Medical and Chemical Expertise in English Trials for Criminal Poisoning.

⁷⁴ Shapiro, B., *Probability and Certainty in Seventeenth Century England*, Princeton, NJ, Princeton University Press, 1983.

Prima facie, considering my figures and the number of guilty verdicts, 54%, it would appear that juries were often hesitant in reaching a guilty verdict in poisoning cases and the distrust of forensic proof is certainly a factor in this hesitancy.⁷⁵ Of note is that in the thirty-four cases where the accused were found guilty only eighteen (53%) were executed.⁷⁶

Until the twentieth century it could perhaps be said that there just did not exist reliable and forensically useful information. Thus, with the many lacunae in materials, and deficiencies in research of former times, it is hardly surprising that so many juries permitted poisoners to walk free from court or indeed that those who poisoned were simply never brought to justice. Poisoning was, based on the patterns of the cases presented, likely to have been widespread among the lower classes of nineteenth century Scotland. Although a jury of fifteen ordinary persons, ignorant of both medical and legal matters, was perhaps a committee of insufficient expertise to

⁷⁵ $34/63 \times 100 = 54\%$.

⁷⁶ $18/34 \times 100 = 53\%$. Often there would be no death penalty, even when a guilty verdict was reached and in particular for the poisoning of children with opium.

compensate for the limits of forensic medicine during this period; legal records can not ever be supposed to present an accurate picture of the true extent to which those wronged, those desperate and destitute, let alone those simply criminal, resorted to the widely available modes of poisoning in the past.⁷⁷

*“And the vitriol madness flushes up in the ruffians’s head,
Till the filthy by-lane rings to the yell of the trampled wife,
And chalk and alum and plaster are sold to the poor for bread,
And the spirit of murder works in the very means of life.
And sleep must lie down arm’d for the villainous centre bits,
Grind on the wakeful ear in the hush of the moonless nights,
While another is cheating the sick of a last few gasps, as he sits.
To poison’d poison behind his crimson lights.”⁷⁸*

⁷⁷ It is the opinion of the writer that juries today can be a hindrance to justice and that perhaps the correct way forward for justice in this country are trials without juries. See for example *Daily Record*, Saturday, April 12th, 2003, where it was reported that during The High Court trial of Starrs, 22, of Fintrayside, Dundee for assaulting a 13 month old boy to his severe injury and to the danger of life, two members of the jury were discharged. The first juror was discharged for being drunk and the second juror, discharged as she had personal knowledge of the case. A further third member of said jury came close to being held in contempt of court due to her drinking during the trial.

⁷⁸ Tennyson, Alfred, Lord, *Maud*, (1855), Part 1, Verses, X & XI, Lines 37-44.

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